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Publication in the JOURNAL OF PERIODONTOLOGY *is not to be understood as endorsement by the Journal or the* AMERICAN ACADEMY OF PERIODONTOLOGY *of opinions expressed in any article, abstract or extract. The object of publishing the various views is to present them without prejudice to the reader for his own evaluation.*

THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health. The plan to continue the publication of abstracts indefinitely will make the Journal a fund of extensive information, readily available to students, teachers, researchers, and clinicians.

The Journal is the official organ of the American Academy of Periodontology and will contain, therefore, those papers of scientific and clinical interest which are presented at the annual meetings of that society. Abstracts of important articles on periodontia that appeared during the year of 1941 are published in this number.

We are grateful to all who have made available the subject matter from which the abstracts and copies of abstracts have been made, including the authors of the original articles, the owners of the journals mentioned in the text of the abstract section, and those who have aided in any way in compiling the material comprising this issue.

THE MEMBERS of the American Academy of Periodontology were informed by letters mailed July 3, 1942, that the Twenty-Ninth Annual Meeting scheduled for Boston, August 20, 21, 22, 1942, had been indefinitely postponed at the suggestion of Mr. Eastman, Coördinator of Defense Transportation. An excellent program had been arranged (See pp. 99 and 118). While the announcement was made and received with regrets, this small sacrifice is made most willingly to promote the war effort for the defense of all American institutions.

THE JOURNAL OF PERIODONTOLOGY

JULY, NINETEEN HUNDRED FORTY-TWO

Periodontology in Dental Education*

By HAROLD J. LEONARD, D.D.S., B.A., *New York City*

PERIODONTOLOGY is the sum of knowledge concerning the teeth in relation to their investing and supporting tissues and structures. Periodontia is the art of treating the disorders and maintaining the health of the teeth in relation to their investing and supporting tissues and structures.

The disorders embraced in this field account for about half of the teeth lost by civilized man and by far the majority of the teeth lost by individuals who seek dental care in order that their teeth may be maintained. Careful dentists are pretty well able to maintain the teeth from loss due to caries and its consequent pulpal disease in patients who will coöperate by seeking regular examination and treatment during their lifetime. They are largely failing with these same patients to save the teeth from loss due to periodontal disorders. So large a proportion of people lose their teeth before old age, that it has been assumed by the laity and even by dentists that toothlessness, like baldness and other senile defects, is a normal accompaniment of old age.

The loss of teeth due to periodontal disease is no more inevitable than their loss due to caries. Hundreds of dentists who have educated themselves in periodontology and trained themselves in periodontia are maintaining the teeth of their patients in periodontal health throughout their lifetime. In the hands of such dentists, the loss of a tooth due to periodontal disease is exceptional. Such a loss represents to the periodontist a failure in his diagnosis and treatment, or in the coöperation of the patient. Although there are factors in diagnosis and treatment which the periodontist recognizes as still unknown, the dentist who is proficient in this field knows enough for failures to be extremely rare.

The reason why a majority of conscientious dentists are failing unnecessarily in the periodontal field is because they have not been educated in periodontology or trained in periodontia in their dental schools. When I was graduated, in 1912, there were but two schools, so far as I can learn, that gave organized courses in "pyorrhea treatment" as it was then called. These were in the University of Toronto, under the late Andrew J. McDonagh, and in the University of Minnesota, under Thomas B. Hartzell. At present all but three of the American schools ostensibly give such courses, but in nineteen only, of the forty-six schools, are the courses given by teachers who could by any stretch of the imagination be

* Read before the Post-Graduate Study Group of the American Academy of Periodontology, at the W. K. Kellogg Institute, Ann Arbor, Michigan, May 19, 1942.

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considered competent periodontists. And even in these nineteen, the amount of time allowed is in several instances too low for adequate presentation and training in the subject. In several of the nineteen a lecture series only is given by a periodontist while the important technical training is left for the clinical staff who are in no sense prepared for such teaching.

A great error, in my opinion, was committed by the Curriculum Survey Committee of the American Association of Dental Schools when it recommended in its report of 1935, that "treatment of diseases of the gingivae and investing tissues of the teeth" be made a subdivision of "Oral Medicine" of which "Diseases of the dental pulp and apical and periapical tissues" and "Diseases of the other tissues of the mouth which do not require surgical treatment," are coördinate subdivisions. The outline of subject-matter and the time allowance for instruction as recommended are not too bad. The error is in grouping subjects together which do not naturally fit together. It is conceivable that a dentist might be found who has made himself expert in the diagnosis and treatment of pulp, root canal, and periapical disorders, of soft tissue lesions of the mouth, and of gingival and periodontal disorders so that he could teach or head a division of oral medicine. But such persons are rare indeed. If the dental pathological phases of these subjects were all that mattered, it would not be so difficult. The Curriculum Survey Committee evidently viewed them in this light, as all branches of clinical oral pathology. Such an approach is purely theoretical. As a matter of fact, periodontia cannot be taught adequately by one who is not himself a competent periodontist. The fact is demonstrated by the poor showing on periodontia cases made by otherwise excellent dentists who have graduated from institutions in which periodontia is taught by teachers whose background is oral pathology and not clinical periodontia. Periodontology is based on pathology but periodontia is no more a branch of pathology than is orthopedic surgery. Orthodontics is based on several fundamental sciences, but no one would think that a good orthodontics course could be taught by a scientist who was not himself a competent experienced orthodontist.

Another stumbling block in the teaching of clinical periodontia is the artificial distinction between the so-called dental prophylaxis and periodontia. In teaching prophylaxis, the student is first taught on manikins to polish lead pencil markings from manikin teeth, then to remove artificial calculus glued to them. After a few sessions devoted to these technics the student starts his clinical experience with patients at the chair by removing deposits and stains from the exposed surfaces of the teeth and polishing the teeth. The instruments he is furnished for this work are not adapted for the removal of subgingival deposits. The subgingival deposits unless visible are simply not removed. Many of the patients coming for dental prophylaxis treatment are children and young people with no periodontal pockets. Yet they often have fairly deep gingival sulci on the first molars with considerable subgingival calculus in them. Such calculus is the sequel of a gingivitis and lack of hygiene at an earlier period. If these deposits are not removed, the sulci will gradually deepen into pockets. In spite of regular prophylactic treatments, the time will come when dentist and patient suddenly awaken to the fact that considerably advanced periodontoclasia is present. The patient then becomes a periodontia case.

The student at the outset of his clinical practice should be trained to clean the teeth to the line of gingival attachment whether the sulci are shallow or have deepened into pockets. A prophylactic treatment that fails to remove all subgingival deposit is worse than useless from a health standpoint because it gives the patient a feeling of confidence that he is getting preventive treatment when in fact he is not. The removal of visible stains and debris without removal of the insidious subgingival deposits is a cosmetic procedure, certainly not an important health measure.

The student should have his set of periodontia instruments at the beginning of his work at the chair in the sophomore year. He should be taught the removal of subgingival deposits and the home care methods which his patients require. The only difference in the procedure for the sophomore, junior, and senior periodontia clinic courses is that in so far as possible cases without periodontoclasia are selected for the sophomores while those with advanced periodontoclasia are selected for the upper classes. The charting and diagnostic procedures should also be at a

minimum in the sophomore year and very thorough in the upper classes. The clinical work should all be periodontia with no part called prophylaxis. Such preventive measures as polishing, home care, smoothing filling margins, burnishing and polishing superficial enamel caries, and instruction in diet should be an integral part of the course as it goes along—not a separate division of the subject. In the field of periodontology no good but much harm comes from any tendency to separate preventive and therapeutic procedures into distinct subjects.

Periodontology is not simply concerned with bacteria, deposits, gingival congestion, and systemic low resistance to infection. It is also greatly concerned with function. The strength and health of the periodontal membrane and supporting bone are dependent on normal and vigorous masticatory function. Lack of function leads to degenerated periodontal membrane which may have several bad effects. Abnormal function in which the tooth receives an excessive back and forth movement from which it cannot shift to a position of stability is almost if not quite as important a cause of periodontal disease as faulty hygiene.

Treatment for such abnormal function is in most cases restoration and replacement procedures—fixed bridgework, inlays for contact, partial dentures. Reshaping by grinding of teeth so as to fit them into a smooth well-functioning articulation is only a small part of such treatment. A large portion of the treatment of periodontal disorders consists of procedures taught in the operative and prosthetic departments. Conversely, a major reason for making fixed bridge and tooth-borne partial dentures is the prevention and relief of injurious strains on the periodontal tissues which would otherwise lead to periodontal disease and tooth loss. Even orthodontics, except for its cosmetic aspects, is largely for the purpose of arranging the teeth so that periodontal disease will be prevented.

Periodontology is not a little subdivision of oral medicine like soft tissue lesions; it is the very framework of dentistry. Outside of diseases of the strictly dental tissues and dental technics, the diagnosis and treatment of dental defects are pretty much in the field of periodontology. How can any dental school be recognized as teaching dentistry when no more attention is given periodontology and periodontia than is covered in the textbooks of oral pathology? The lack of sound education, training, and emphasis on periodontology in dental education means a distorted and imperfect conception of dentistry that must lead graduates to a very inadequate practice. Perfection in the technical procedures of operative and prosthetic dentistry will not save the teeth when not guided by a sound knowledge of their periodontal implications.

A great deal is heard in dental educational circles of the gulf between the fundamental biological science subjects and the clinical subjects of dentistry. The student takes extensive courses in anatomy, pathology, and physiology, and when he completes them that is the last he hears of them excepting for state-board examinations. Everyone gives lip service to the idea that dentistry is based on these sciences but actually very few schools are able to make satisfactory application of them in the clinical subjects of dentistry. Leaving out oral surgery, the main clinical subjects are operative dentistry, prosthetic dentistry, and orthodontics, which in most schools are largely technical departments devoted to mechanical manipulative procedures. The solution to the problem of dovetailing the fundamental sciences with clinical dentistry lies in the teaching of oral diagnosis, oral hygiene, and periodontology. It also lies in such close integration of these subjects and operative and prosthetic dentistry that each subject enhances the others by contributing facets to it.

Oral diagnosis and treatment planning are so closely linked with periodontology both in teaching and practice that they must be specially considered. The Curriculum Survey Committee did much better by Oral Diagnosis than by Periodontology in its report: *A Course of Study in Dentistry.* Oral Diagnosis was presented as the trunk of the tree of dental practice, with its roots in the fundamental sciences and its branches in the technics of dental procedures. Twenty-nine class hours are recommended and one hundred and twenty hours of clinical practice. The class hours are devoted to methods for the collection of data; oral diagnostic technics, abnormalities of the mouth, physi-

cal examination of the patient, clinical pathological tests, significance of pain, signs and symptoms of disorders of the various oral structures—hard tissues of teeth, pulp, periapical tissues, gingivae and periodontal mucous membrane, dentition, mandible and maxillae, maxillary sinus, salivary glands, systemic diseases related to dental conditions, etc. The clinic hours are devoted to examination at the chair, radiographic and laboratory examinations, the making of a diagnosis, prognosis, and provisional plan of treatment. In practice treatment planning usually consists of noting cavities to be filled, teeth extracted, spaces to be bridged, prosthetics and periodontia indicated, presumably leaving it to the respective clinical branches to make more detailed plans. Of the twenty-nine class hours, two are devoted to "signs and symptoms of disorders of the gingivae and peridental tissues." These cover recognition, significance, and record of the following: pain, tenderness, swelling, heat, hemorrhage, presence or irritants (mechanical, chemical, bacterial), odor of the breath, motility of the teeth, surface ulceration, disturbed occlusion, recession, drifting of teeth, color, formation of pockets, abnormal depth of gingival crevice, subgingival suppuration, calculus on the teeth, thickened margins, density, presence of faulty contacts, dryness, dull percussion note, fistulae, necrosis, rounded interdental papillae, report on radiographic examination, alveolar resorption, report on laboratory examinations, systemic disturbances that may be predisposing causes.

This is all excellent so far as it goes. It does not go far enough, however, so that the student realizes from it how local irritants are causing disease to the periodontal structures and how the treatment must be planned in detail to eliminate these local irritants and prevent their recurrence. It is not enough in the treatment planning to note that periodontia and prosthetics are indicated.

The theory course in treatment of diseases of the gingivae and investing tissues of the teeth as recommended by the Curriculum Survey Committee fails to carry on from there. The theory course of fifteen hours deals mostly with these diseases from the standpoint of oral pathology. Twenty hours of clinic time are recommended for oral prophylaxis while one hundred and eighty to two hundred hours are given to oral medicine, including the diagnosis and treatment of (a) diseases of the pulp and apical and periapical tissues, (b) the gingivae and investing tissues of the teeth, and (c) the other tissues of the mouth which do not require surgical treatment. The course in prosthetic dentistry as suggested by the Curriculum Survey mentions something about the biological basis of the subject but in detail is devoted to technical procedures.

The point I am attempting to make is that treatment planning based on consideration of the supporting structures of the teeth is lost in the shuffle of courses even in the model curriculum suggested by the Curriculum Survey Committee. Needless to say it is much more lost in schools where there is often no one with practical knowledge, skill, and experience in periodontia. Because of lack of such knowledge, skill, and experience, there are deans of our dental schools today who regard periodontoclasia as incurable and who have been heard to state that involved teeth will sooner or later be lost no matter what is done. Schools dominated by such deans naturally do not have good periodontia teaching.

Three factors are chiefly responsible for the hopeless attitude toward periodontal diseases assumed by those who have not been educated and trained to understand and treat them. The first of these is lack of skill in removing irritating deposits from the subgingival tooth surfaces. The second is lack of understanding of the use of massage in restoring deep congested periodontal tissues to normal circulation and health. The third is the faulty relationship between periodontia and operative and prosthetic dentistry.

Operative and prosthetic procedures should be designed so that the teeth are stabilized, put into non-irritating function, and prevented from developing undue strains and stresses. A prosthetic appliance which causes or permits the teeth to be lost through its use is much more of a failure from a dental standpoint than a well-designed appliance of such poor workmanship or material that it breaks or wears out prematurely. The design of dental prosthetic appliances and to a considerable extent the

shaping of dental restorations is a biological problem in the field of periodontology. The closest relationship should exist between the clinical divisions of oral diagnosis, periodontia, operative dentistry, and prosthetic dentistry.

A teaching institution in which oral diagnosis only notes dental defects, periodontia only cleans the teeth and teaches home care, operative dentistry only fills cavities, and prosthetic dentistry only replaces missing teeth with sturdy and beautiful bridges and dentures is inadequate no matter how good the technical teaching. The correlation of these subjects so as to lead in each case to the establishment and maintenance of health, function, comfort, and beauty is essential. The correlating factor is periodontology.

The intimate relationship between periodontology and other branches of clinical dentistry makes me feel that it is a mistake to regard periodontology as a specialty of dentistry that should be conducted as a limited practice like oral surgery or orthodontics. In my opinion effort to segregate periodontia from general practice is wrong theoretically and practically. A general practice based on inadequate knowledge of periodontology and skill in periodontia treatment is inadequate and in many cases will fail to maintain the teeth of patients in health throughout the lifetime. Conversely, a practice limited to treatment of gingival and root surface tissues and to establishment of hygiene in the mouth, without control of operative and prosthetic procedures, is inadequate and will also lead to failures in maintaining the teeth in health throughout the lifetime. The two must be united in the practice of dentistry. Periodontology can no more be segregated from such practice than can oral diagnosis or operative dentistry.

This is not to say that there is no need for dentists who have become expert in knowledge of periodontology and skill in periodontia to serve as consultants and for reference in difficult cases. There should be those who by reason of interest, study, and special talent become outstandingly proficient in this field while others do the same in full-denture prosthesis or porcelain work or other divisions of clinical dentistry. Such men should serve as teachers, clinicians, and expert practitioners to whom patients may be referred for special service. It is quite right that those who have such special interests should have societies for the promotion of the science and technics in their fields. It is also proper that there should be examining boards in the special fields to certify those who are in fact expert so that they may be known to those who need to consult them. There should also be postgraduate courses in the universities so that those who wish to get special education and training in any of these divisions of clinical dentistry may do so. It is also satisfactory for dentists in group practice to limit themselves to certain branches of clinical practice as long as the patients obtain total dental service from the group. The error occurs when periodontology is regarded as something that may be safely separated from clinical dentistry as orthodontics and oral surgery have been. Or, in dental teaching as treatment of pulp and periapical lesions, and treatment of soft tissues of the mouth may be regarded.

Periodontology and its related oral diagnosis and treatment planning are the mind of dental practice. Procedures in periodontia, operative dentistry, prosthetic dentistry, and to a lesser extent orthodontics, are the hands by which the mind operates. The aim of dental practice is the establishment and maintenance of health, function, comfort, and beauty in the mouths of those to whom we minister. Dental practice without periodontology is hands working without a mind.

While giving no less emphasis on excellence in the technics, the great need in dental practice is a basis of periodontology. The establishment and maintenance of health, function, comfort, and beauty in the mouths of those to whom we minister is the function of clinical dentistry. This aim cannot be carried out unless all procedures are closely correlated. The failures of dentistry are largely those of the supporting structures. Such failures are with few exceptions unnecessary. Present knowledge to prevent them is available in the science of periodontology. Such knowledge must be combined in the practice of clinical dentistry. It is the job of the dental schools so to combine it. Periodontology is not a subdivision of oral pathology. It is an essential part of dental science and practice and must be given the weight and substance due it in the education of dentists.

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The Tooth-Supporting Apparatus Under Occlusal Changes*

By CARL BREITNER, M. D., D. D. S. *New York City*

Periodontia and orthodontia are closely related fields of dentistry. This relationship may not be obvious at first glance because the periodontist's primary interest is in the stabilization of teeth, while the orthodontist is most often interested in moving them. The orthodontist may, however, very well become interested in tooth stabilization when he attempts to change the relation of the jaws, in other words, when he attempts, instead of moving teeth, to move the jaws themselves. He then finds himself in urgent need of strongly anchored teeth which will not yield to those forces which tend to shift their position. The periodontist, on the other hand, must often alter the load borne by the teeth when he makes artificial restorations, and he may also be confronted with migration of teeth due to periodontal disease. He then becomes interested in shifting teeth. You see, therefore, that the interests of periodontists and orthodontists frequently meet, especially when it comes to gauging the probable resistance and reaction of the tooth-supporting apparatus to forces which are applied to the teeth.

Before discussing some of the reactions of the tooth-supporting structures to forces applied to the teeth, let me suggest a general conception of the masticatory apparatus. This conception then will offer a basis for our understanding of the reactions which may or may not be expected under different conditions.

The fully developed masticatory skeleton is kept in its relatively constant shape by the interplay of various forces, most of which originate from muscular tissue. (See Figure 1.) A portion of these forces are attached directly to the bone, as are the masticatory muscles; partly they form a sort of envelopment—a wrapper around the skeleton—as do the facial muscles, and partly they act from the inside of the structure—as does the tongue. There is also a group of forces which do not originate from muscle cells, but rather from growth impulses which, in varying degree, are present during the entire life. Such a force is the tendency of the teeth to erupt. There exist additional forces some of which are listed in the Table following. *To maintain the shape of the skeleton, all of the forces must counteract one another in perfect balance.* Any disturbance of this balance will cause bone transformations which will continue as long as the balance is not reëstablished. I have called this balance Functional Equilibrium (F. E.) (1).

TABLE. SOME ANTAGONISTIC FORCES ACTING UPON THE MASTICATORY APPARATUS

Lips →	← Tongue
Cheeks →	← Tongue
Eruption (growth of teeth) →	← Masticatory muscles (masseter, temporalis, internal pterygoid)
Air pressure on skin and nasal cavity →	← Tongue in closed mouth, air pressure in open mouth
Masseter →	← Elasticity of periodontal membrane particularly of molars, and suprahyoid muscles
Internal pterygoid	
a) in vertical movement →	← Same as masseter
b) in lateral movement →	← Internal pterygoid of other side
External pterygoid	
a) in anterior movement →	← Posterior third of temporalis, suprahyoid group, diagastricus, muscles of the neck
b) in lateral movement →	← External pterygoid of other side

*Read at the April 17, 1942, Meeting of the Post-Graduate Periodontia Study Group of Columbia University. *New York City.*

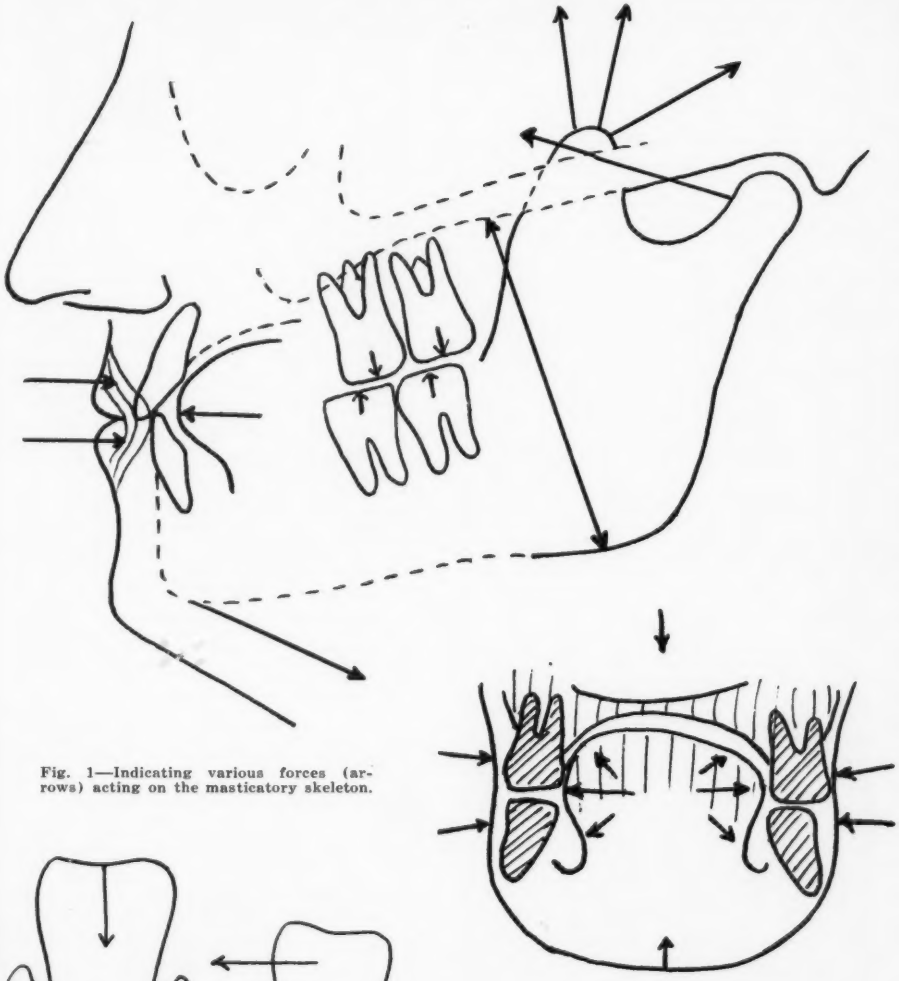

Fig. 1—Indicating various forces (arrows) acting on the masticatory skeleton.

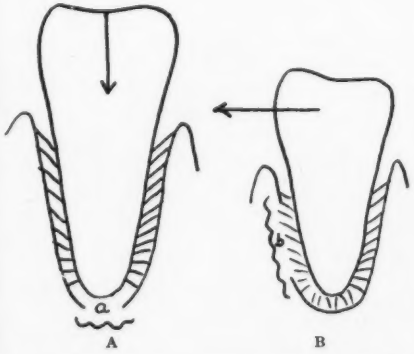

Fig. 2—A—Vertical force—tautens periodontal fibers. Bone is strengthened. Actual pressure only at the apex and in bifurcations. (a) Area of actual pressure only at the apex. B—Lateral force—always causes pressure in vast areas. Bone yields by resorption. (b) Area of pressure.

Both bone, or resistance, and muscle, or forces, may undergo changes during life. Teeth may be lost, which will change the resistance, and muscular forces may be altered by exercise, disease, or artificial changes such as occur in attempted changes of the vertical dimension.

Before discussing this in detail let us consider an important attribute of muscular tissue. It is known to physiologists (Wright (2), Howells (3), Macleod (4)) that the elastic tension of a muscle increases when

a muscle is elongated, and decreases when a muscle is permitted to contract further than to its normal length. We may compare the muscle to a rubber band under tension. There is only a slight but important difference. The tension of a rubber band will increase in arithmetical proportion to its elongation; if we elongate the muscle ten per cent, its elastic pull may be increased much more than ten per cent. In addition to this increased tension which is due to the so-called stretch reflex, W. H. Howells (3) says, about plain muscle elasticity, "A muscle that is in a state of elastic tension contracts more promptly and more effectively for a given stimulus than one which is entirely relaxed." If we raise the bite, increasing the length of certain muscles, we therefore increase their constant elastic pull considerably.

It can be easily seen that any change in the vertical dimension will disturb the balance between the forces acting upon the masticatory apparatus (that is, the F. E.). To avoid misunderstanding, let us agree that we shall understand by "vertical dimension," in the following discussion, the distance between the zygomatic arch and the lower border of the mandible in the molar region. This vertical dimension in a given individual represents a certain determined value. It is the distance at which muscular forces and counteracting resistance balance one another. As we said before, this balance is essential to stability, and any disturbance thereof will cause changes leading to a reëstablishment of the balance. From this point of view a number of clinical observations will now be explained.

ATTEMPTED INCREASE OF THE NORMAL VERTICAL DIMENSION

Starting with a normal individual, attempted increase of the normal vertical dimension will mean elongation and therefore increased pull of internal pterygoid, masseter, and temporalis muscles. Since this increased pull is not counteracted by increased force of the antagonistic muscles, or by any other considerable increased resistance, there will be a disharmony, or a disturbed F. E. This situation cannot prevail, and changes will occur leading to reëstablishment of the original balance. Bone, as the more plastic tissue, will yield to muscle (5) (6). Histologic specimens gained from experimental material subjected to a disturbed F. E. show bone changes in various regions, such as in the alveolar bone, in the temporomandibular articulation, in the region of the angle of the mandible, etc. (1) (7) (8). In other words, because of the tendency of the original F. E. to reëstablish itself, a *permanent artificial increase* of the vertical dimension beyond its normal value is *probably impossible.*

ATTEMPTED DECREASE OF THE NORMAL VERTICAL DIMENSION

Decrease of the normal vertical dimension is made possible either by increasing the muscular forces (temporary bite-raising in molar region) or by decreasing the counteracting resistance, that is for instance, by extraction of a few molar teeth. In the latter case the ensuing closure of the bite is naturally accompanied by decrease in length of mouth-closing muscles which thereby lose tension. This loss of tension then will at a certain point balance the reduced resistance of the mutilated skeleton. In other words, at a new decreased vertical dimension a new equilibrium will be established between the weakened bone and the weakened muscles. Therefore; *artificial* decrease of the vertical dimension, for instance by mutilation, is *possible.*

ATTEMPTED INCREASE OF A PREVIOUSLY COLLAPSED BITE

Increase of a previously collapsed bite is made possible by use of certain procedures. If the bite has collapsed e. g. through loss of teeth, and we raise it by increasing the length of the remaining teeth (for instance with caps) it would return to the closed level. But if we put in artificial restorations of the *missing teeth* we increase the resistance of the skeleton to the muscular pull. An increased muscular pull, which is created by raising the bite to the original level, finds, therefore, its increased, balancing resistance. In other words, *raising the bite in a collapsed case* by prosthetic restorations must be *possible.*

Do we have a criterion for the vertical dimension normal for a given individual? According to present literature, such a criterion does not exist, but there are certain signs which indicate previous closure of the bite, and there are some suggestions as to how to determine the proper vertical dimension in edentulous cases. Signs indicating a closed bite are: rolling out of the lower lip,

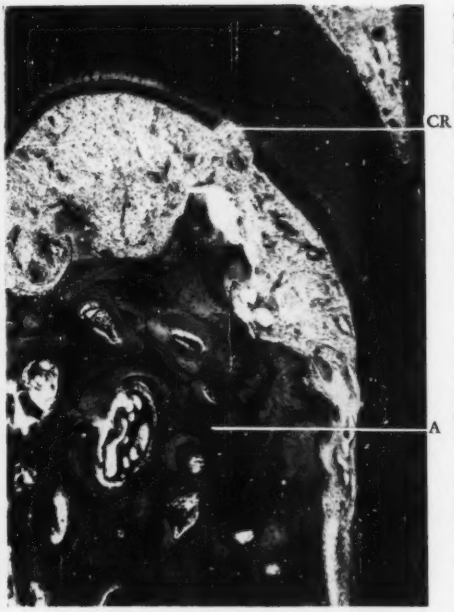

Fig. 3—Bifurcation of a monkey molar which had been under increased vertical stress for three weeks (Cap). Note density of alveolar bone (A), widening of the periodontal space under the bifurcation. There is a small area of cement resorption (CR). Compare with Fig. 4 showing analogous area in a normal monkey.

and creation of a groove below it, fanning of the upper incisors and loosening, or tilting, of molars. Interesting among the suggested aids in determining the proper vertical dimension is Boos' (9) suggestion to determine with a gnathodynamometer the vertical dimension at which the reading is the highest. According to all indications the greatest force in closing the mouth can be exerted at approximately that level.

How and where, then, do the changes occur which lead to a reëstablishment of the functional equilibrium? Histologic investigations on monkeys have shown that bone changes occur in a number of areas. We may divide them into intra-alveolar and extra-alveolar changes. By intra-alveolar changes we mean all the changes which take place within the alveolar process. All investigators agree that these changes, namely bone resorption and bone deposition, do occur, and as you know the intra-alveolar changes form the basis for all orthodontic treatment or alignment of teeth. But there are also changes which occur in so-called extra-alveolar areas, that is, beyond the alveolar processes. After experimental treatment such changes could be detected in the condyle of the mandible and in the glenoid fossa of the temporal bone, in other words, in the surroundings of the temporomandibular articulation. After alterations in the vertical dimension they were found especially at the lower border of the mandible, the ramus, and at the mandibular angle. Such changes have been described and illustrated in various articles of the author (7) (8) (1).

Whether intra-alveolar or extra-alveolar bone changes are preponderant is determined by the degree of yielding of the tooth-supporting apparatus. That is, if it is the tooth-supporting apparatus which yields, the extra-alveolar bone changes will be negligible, and vice versa. Therefore, it becomes important to predict the reaction of the tooth-supporting structures in any prosthetic or orthodontic procedure. The reaction of these tissues to a force is influenced by a number of factors, such as the intensity of the force, the method and location of its

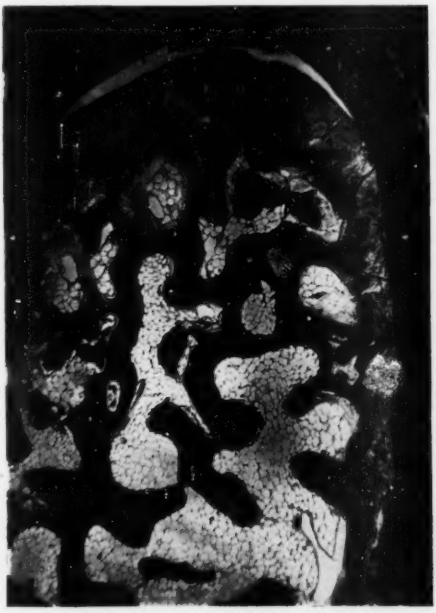

Fig. 4—Bifurcation of a monkey molar under normal conditions. (Same magnification as Fig. 3.) Note normal density of alveolar bone.

Fig. 5—Apex of molar shown in Fig. 3. Increased vertical pressure has produced resorption of cementum and dentin (R). There is repair by cementoid tissue taking place at (C). Compare with Fig. 6 showing an area along the root of the same molar. B: Bone, P: Periodontal membrane.

application, length of the clinical root, and others. One of the factors greatly modifying the reaction of the tooth supporting apparatus to a force is the *direction in which the force is applied.*

There is a basic difference between the influence of a force applied in the direction of the longitudinal axis of the tooth, called "vertical force," and the effect of a force applied to a tooth in any other direction, called "lateral force." The vertical force, up to a certain limit of course, does not cause much resorption but rather causes strengthening of the alveolar bone through newly formed bone tissue. The lateral force will always cause bone resorption on the side of pressure and deposition on the side of pull. This characteristic reaction, uniformly found in histologic investigations by many authors, including Gottlieb and Orban (10), Orban (11), and MacMillan (12), is probably due to the fact that the tooth-supporting apparatus is better designed to withstand forces in the vertical direction. Due to the suspension of the tooth in the socket, a vertical force does not necessarily produce increased pressure within the periodontal space leading to resorption, whereas, a lateral force always produces increased pressure in large areas. (See Figure 2.)

This variation in the reactions of the periodontal tissue to forces in different directions assumes significance for the periodontist whose task frequently is to establish an occlusion in which lateral forces are eliminated as far as possible. Let us therefore consider forces in various directions from this point of view.

Histologic specimens obtained from animal experimentation and human material reveal the following:

FORCES PARALLEL TO THE LONGITUDINAL AXIS—VERTICAL FORCES

Other factors being equal, vertical forces will cause the least bone destruction. There will be scattered places in the periodontal space where the bone is resorbed under the influence of direct pressure (apex and bifurcation), whereas, in the vast majority of areas the pressure is transformed into tension due to the characteristic arrangement of the fibers of the periodontal membrane. This may explain the frequently observed increase of density of the alveolar bone under such influence. (Figures 3, 4, and 5.) Actual pressure is to be found only in the bifurcation and at the apex, while along the entire length of the root we may even find deposition of new bone. (Figure 6.)

FORCES ACTING AT AN ANGLE TO THE LONGITUDINAL AXIS—LATERAL FORCES

Lateral forces, up to a certain limit, will have the familiar effect on the alveolar bone of resorption on the side of pressure, and deposition on the side of pull. In this way a new socket is formed, and the tooth, sometimes including the alveolar process, is shifted from one area to another. It is these lateral forces which are used in almost all orthodontic appliances in the well-known attempt to shift teeth. Under favorable conditions bone destruction and formation balance each other approximately, and the tooth is not much loosened. But, however weak the lateral force, the tooth will react to it with migration, and extra-alveolar bone transformations to a considerable degree cannot therefore be expected.

COMBINATION OF LATERAL AND VERTICAL FORCES

A combination of lateral and vertical forces places great strain on the tooth-supporting tissues, as can be observed in many specimens (7). Beyond a certain limit the combination proves deleterious to the alveolar bone. Excessive destruction of bone and cementum, particularly in the apical areas and bifurcation of molars is found under these circumstances. (Figure 7.) The periodontal tissues, cementum, periodontal membrane, and alveolar bone are crushed and undergo grave pathologic changes. Those changes may heal, particularly in young and resistant individuals. It is this combination of vertical and lateral forces, however, which often is found to play havoc with the tooth-supporting apparatus. It should therefore be avoided whenever possible, particularly in cases of periodontoclasia.

Steep cusps and deep grooves in the occlusal surface usually produce this dangerous combination of forces. In cases of lowered resistance this fact alone may cause a loosening of teeth, not to mention the danger of raising the bite in this way. The combination of lateral and vertical forces is also often produced by the dentist in constructing artifical restorations with clasps or attachments to the remaining teeth.

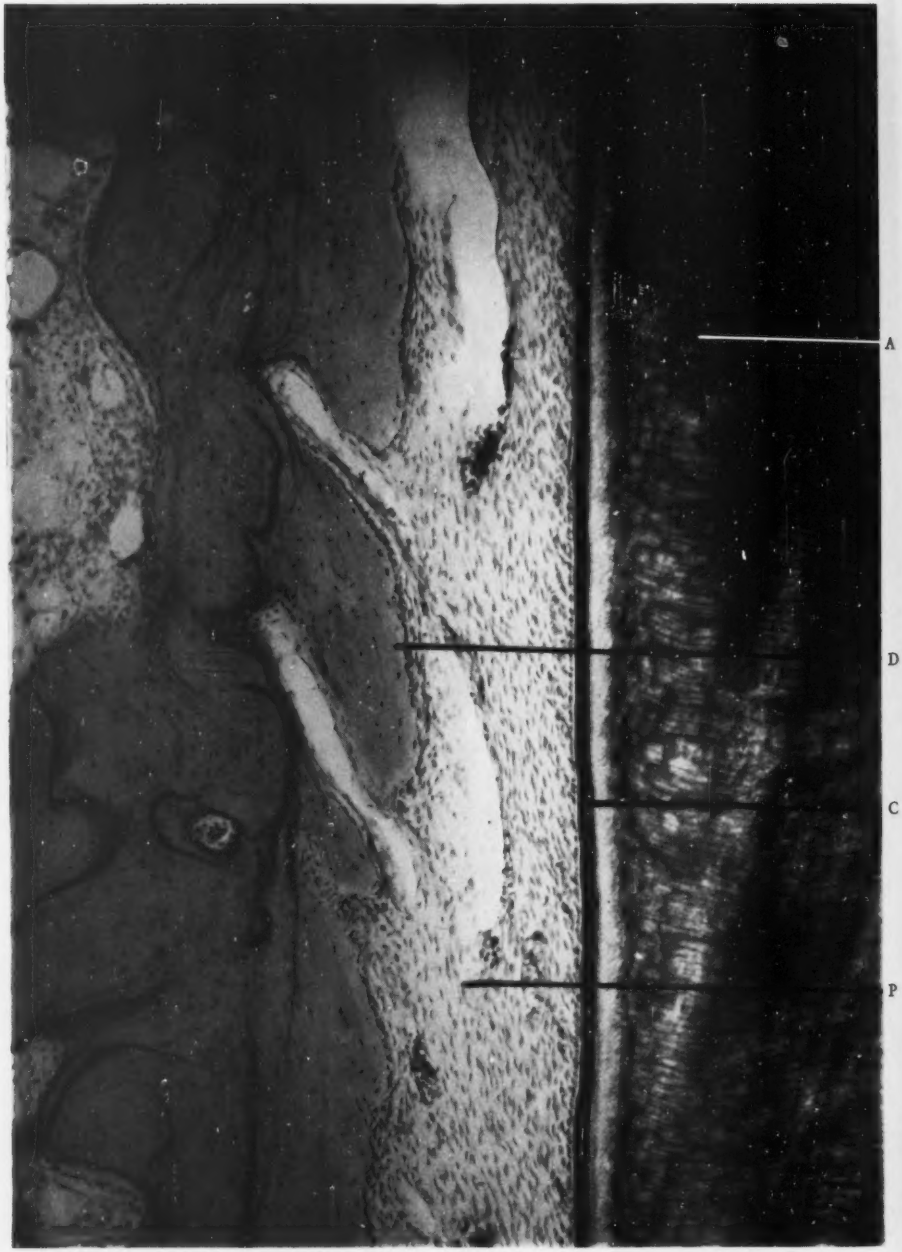

Fig. 6—Area along the root of molar shown in Figs. 3 and 5. Note deposition of new bone (D) which is arranged in a downward direction according to the vertical pressure on the tooth. A: Dentin. C: Cementum. P: Periodontal membrane.

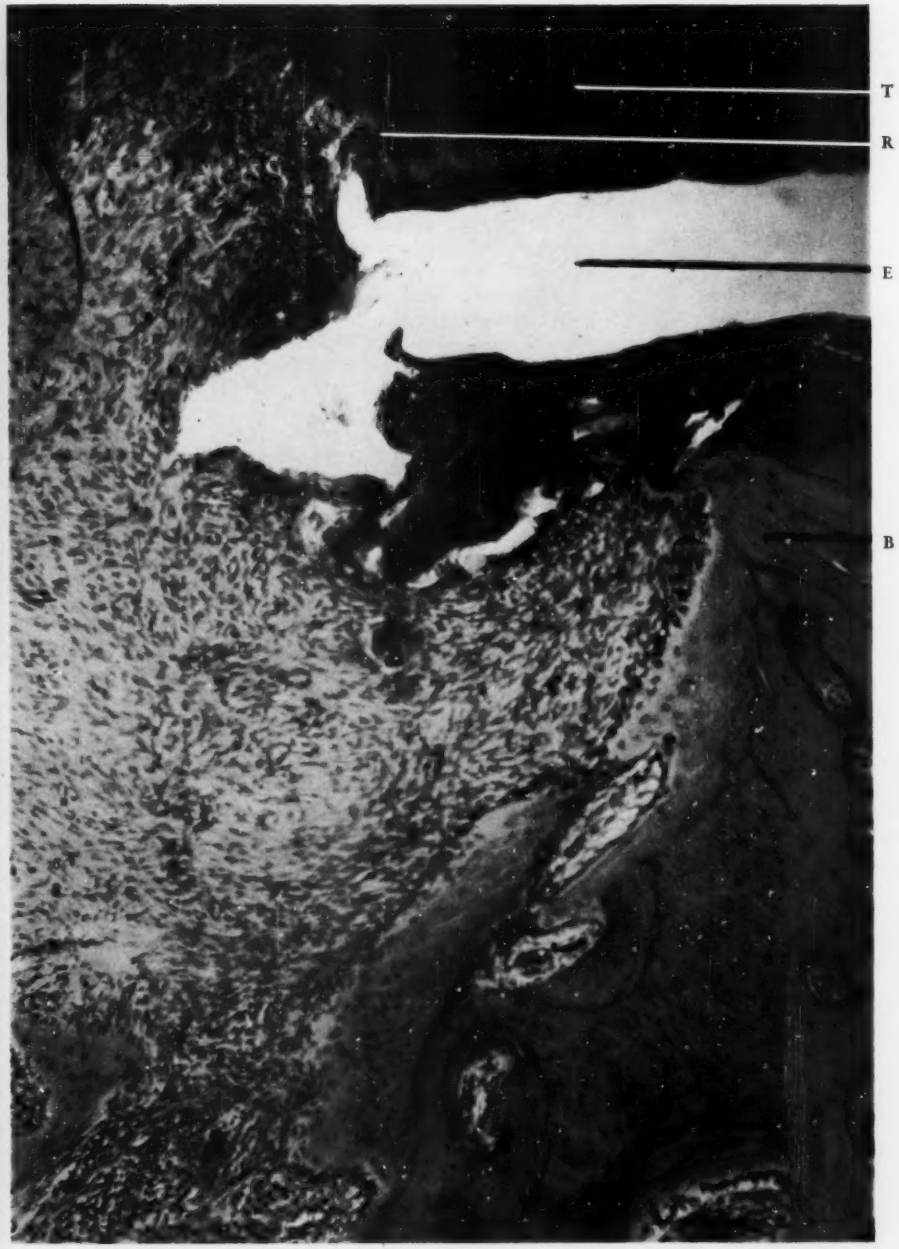

Fig. 7—Bifurcation of a monkey molar under continued lateral and vertical strain. Note destruction of all tissue between tooth (T) and bone (B). There is an empty space at (E). The continuity of tooth and bone can be reëstablished only by "back resorption" of the entire area. Note, therefore, extensive resorption of dentin at (R).

CONCLUSIONS

From histologic findings, and from clinical experience, we may deduce that natural abrasion of the teeth is a protective factor against damage to the tooth-supporting apparatus because it reduces the forces acting laterally on the teeth. Since vertical forces, toward the base of the socket, are naturally always present, the elimination of lateral forces implies the elimination of that dangerous combination of vertical and lateral forces. Purely lateral forces of a limited intensity, such as are used in orthodontic appliances may be applied in patients of any age, as long as they are used in a dentition without marked pathologic changes.

Vertical forces such as those used in bite planes, caps, and splints may be applied in dentitions with normal periodontal tissues. If used judiciously, and constructed without interdigitations, they will produce changes in the occlusion mainly by inducing extra-alveolar bone changes.

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Item of Interest

Members of the Academy will be pleased to know that Dr. R. Gordon Agnew has arrived safely in China to continue his work at West China Union University at Chengtu, Szechwan Province, near Chungking.

In Memoriam

ANDREW J. McDONAGH, February 10, 1942

WILLIAM Y. HAYDEN, March 13, 1942

LUTHER A. NEIL, June 17, 1942

CLYDE C. SHERWOOD, July 5, 1942

Principles of Treatment Evolved from Experience and Observation*

By WALTER LEABO, D. D. S.
Shreveport, Louisiana

FEW DENTISTS are qualified to do scientific research. Early training, associations, environment, and a vocational bent enter into the development of research men. Comparatively few of us are so situated as to be able to devote the necessary time, nor do we have the essential background for this type of work. To research men we owe a great debt of gratitude. Those of us engaged in daily efforts toward the elimination and prevention of periodontal disease rarely ever pause to contemplate the chaotic condition of such practice twenty to twenty-five years ago. One attempting or offering to treat or control "pyorrhea" at that time was considered by many of his brother dentists almost in the light of a charlatan. Today, thanks to those devoted men of research, and to the practical application of sound principles by men interested in periodontia, we now occupy a stable and increasingly important position in the science of saving teeth from degenerative disease. It is interesting however, to recall that in the past such men as Patterson, Sarrazin, Smith, and others made their contributions to periodontology by years of hard work, common sense, and observation of nature's assistance, and response to the elimination of irritation. We cannot help but be impressed by the fact that modern research in histology and pathology further establishes the fundamental practices of these men. Not all of our progress can come from the laboratory, nor can we make headway by following blindly each new method or technique which professes to be the only procedure for successful operation. Dogmatic assertions can hardly contain all truth. And truth is what we are constantly seeking in the results of patient hours of research in the laboratory and the often discouraging years spent at the dental chair.

The practice of dentistry with its two great destructive degenerative diseases, caries and periodontoclasia, is a far wider field than our predecessors ever dreamed about. In fact it is rapidly approaching that of a philosophy, in that our degenerations are the results of our civilization, our foods, living habits, economic stresses, and the inability of the human body to cope with the increasing demands made upon it. We are fighting a losing battle unless we recognize this status. Dentistry is one of the two great healing professions into whose hands has been placed a responsibility we must not shirk. We have the opportunity to go far beyond the fields of dentistry. It is not only *dental* degeneration with which we are attempting to cope, but every degeneration to which civilization has subjected the human economy. Nature is our best ally, and she never intended that such degenerations should exist if her laws are followed. This covers a phase of etiology which needs a great deal of elaboration, but which cannot be discussed further at this time.

I shall consider chiefly the local factors producing direct tissue destruction. It seems practical to state these simply as irritation of the periodontal tissues, mechanical, chemical and bacterial. These three blend and combine to such an extent that usually all three exist at the same time, although any one of the three may have originated the inflamatory reactions in the tissues. In the practical treatment and prevention of periodontal disease it is essential that these forms of irritation be eliminated as far as possible, and in many cases the degree of success in treatment is directly dependent upon the thoroughness with which this is accomplished, irrespective of the type of operation used. Of all injurious mechanical forces at work in the dental field, traumatic occlusion probably is most universal, and plays the greatest part in the production of the fatal parietal lesions. Its destructive action is one truth firmly established in our conception of periodontal disease. Its correction is fundamental to all treatment. Unfortunately there is a wide variation of opinion as to how this is

*Read before the American Academy of Periodontology, Houston, Texas, October 23, 1941.

best accomplished. The young dentist and all too often the older men have only a vague idea of the proper technique. Our literature is almost silent on the subject. It seems that here is a need we as members of the Academy would do well to recognize. We decry the excessive grinding and mutilation of teeth, yet there is very little in the literature to correct the efforts of general practitioners who recognize the need to do something to relieve occlusal trauma.

It is obvious to observing men that modern living conditions and habits which have developed without thought with regard to their effect upon human tissues, have created a situation in practically all mouths whereby the gingival crevice is in a constant state of attack by the various irritants previously mentioned. Furthermore the routine care of this tissue is not and can never be a matter of perfunctory "scaling." Its exploration and care must be as carefully carried out as the operative procedures upon any deeper or advanced lesions in the periodontal tissues, plus the elimination of trauma and correction of nutritional errors as far as possible.

There are few patients who sit in our chairs for the treatment of periodontal disease who will not bring up the subject of foods and whether diet has anything to do with their condition, and what can be done to correct it. This is often an embarrassing question since so many of our profession have only a vague idea of just what the important and controlling factors are. We know that calcium deficiency, and lack of various vitamins are usually present, but our ideas as to how they are to be corrected are very confused. Furthermore, we often lack the courage, provided we have the conviction, to tell the truth, especially when we know it will frequently be met with ridicule or at least incredulity. Every one who has observed human degenerations realizes that nutrition is in for a radical change if we are to ultimately make much progress in the prevention of dental degeneration. It behooves us to have something definite to say on the subject of diet to the inquisitive patient. At the risk of incurring considerable criticism the following dietary prescription is offered. The first fundamental is to let everything alone which is made of or contains white flour or white sugar. Drink from a pint to a quart of milk each day. Take eight ounces of orange juice or tomato juice or their equivalent in fresh fruit daily. Eat all kinds of fresh meats, eggs, oysters, shrimp and other sea foods with all the fresh vegetables desired; also whole grain cereals. Advise the patient, when choosing a food to ask himself if it was made by nature or by man, and if it was made by man he is pretty safe in letting it alone.

No phase in periodontal practice carries greater responsibility than that of prophylaxis. It is a common custom for dentists to refer to prophylaxis as a treatment for all manner of gingival inflammations and often for the elimination of periodontal lesions more or less advanced. This is a serious error, constituting a total misinterpretation of the term, and often minimizing the importance of careful and frequently prolonged treatment. We should miss no opportunity to impress upon our confreres in dentistry the exact role that prophylaxis occupies, and to discourage the promiscuous use of the term. Prophylaxis is properly applied to only two classes of patients. First those patients (usually very young persons) whose mouths have no periodontal lesions, and second, those who have had treatment for such lesions and the supporting tissues placed in a relative state of health. Where patients present regularly for this service we often have a difficult and laborious task to perform, and one which may call for all the skill and judgment we possess. Every etiologic factor entering into the production of lesions or recurrence of lesions must be observed. It has been estimated that in a mouth of twenty-eight to thirty-two teeth there are from thirty to thirty-four inches of gingival attachment. In the operation of prophylaxis every portion of this attachment must be carefully examined and the fine curette frequently used if inflammation is to be prevented and further tissue destruction controlled. Subgingival calculus often forms and can be detected before there is any visual indication of the inflammatory reaction in the tissues. It is an obvious truth that no matter by what method the gingival attachment has been made healthy, it can be maintained in that state only by very careful elimination of all irritants at definite periods. The skillful use of the subgingival curette is entirely essential. The smallest type of curette is most effec-

tive in this work, and as sharpening reduces the size of the cutting lip it becomes even more effective. Observation indicates that maintaining the ideal, with the entire absence of inflammation, normal crevice depth throughout the gingival attachment, is all too often a task beyond our accomplishment; however, if by routine attention we can prevent further tissue destruction we have certainly justified the procedure. The ability to render comprehensive preventive service for periodontal disease is the very foundation for treatment and eradication of the deeper and more advanced lesions. Obviously, the failure to detect and prevent the early detachments of the periodontal membrane in the routine care of the mouth in the offices of the general dentist is due more often to the lack of skill in the use of the subgingival curette than to any other factor. None of the basic etiologic factors should be neglected, but unless the operator is accustomed to performing careful curettage it is difficult to understand how he can hope to maintain the health of the surrounding tissues of teeth which it is his obligation to protect.

Practically all periodontists and students of dental pathology are agreed as to the major factors in the development of periodontal disease. But there is considerable variation and divergence of opinion as to procedures and operative technique, the object of which should be the elimination of pathology and the prevention of further disease. One principle is common however, in the management of every type of periodontal lesion. The cemental surface must be rendered entirely free of calcular deposits and other debris clinging to it. Whether the detachment is one millimeter or four millimeters the same principle applies. If one can learn to curette a lesion one millimeter in depth, one can learn to operate in deeper lesions just as effectively, and without having to cut away the overlying tissue in order to see what is being done. We can never make much progress in the prevention of periodontal disease if we harbor the idea that it is necessary to view the cemental surface in order to remove calcular deposits and other infection. The prevention of periodontal disease and the eradication of the thousands of slight and ever recurring lesions is dependent upon the elimination of irritation and trauma, the correct use of the tooth brush and the skillful use of the subgingival curette.

In the past we have heard the infiltrated and resorbing interproximal crest spoken of as necrotic, with the idea advanced that it is necessary to curette this area in order to bring about restoration of health and elimination of pathology. Kronfeld said, "The supposition that loss of bone and formation of a pocket must necessarily go hand in hand is not substantiated by the actual examination of the tissues involved." Clinical experience has demonstrated to many men doing careful subgingival curettage that usually the probe or curette will not pass along the cemental surface to the process. Tissue is still firmly attached to the tooth above the alveolar process. If this is broken down or destroyed, needless tissue is sacrificed, and the possibilities of regeneration of bone and conservation of the supporting tissues jeopardized. We have no reason to believe that this tissue has been separated or cut off from its nutritional supply, as in the case of actual necrotic bone which so frequently results in sequestration, nor that it needs to be destroyed by means of the curette or saw. Histologic research has produced no conclusive evidence necessitating such procedure. Observation leads to the conclusion that if irritation is effectively removed, natural and biologic forces of repair are sent forth to such an extent that lesions are not only eliminated but new bone cells laid down or the remaining ones recalcified. This has been the experience of so many engaged in the treatment of periodontal disease that it represents a truth; one that has the most profound bearing upon the prevention as well as the elimination of periodontal lesions.

Whenever there exists a superfluity of unattached overlying epithelium or gingival tissue its excision recommends itself as a simple common sense measure. However, the tendency is often toward excessive and too hasty excision. Certainly there is no reason to excise gingival tissue merely because it is in a state of inflammation or even hypertrophy. It appeals to common sense to remove the causative factors of tissue irritation and await nature's response. This is often sufficient. As skill in the use of the subgingival curettes increases it is amazing how few cases require other operative interference.

Where it is evident that some excision is essential it may be most effectively accomplished by the use of either straight or curved cataract scissors. These may be used in such a way as not to injure the sound attachment, but to trim to it. A topical anaesthetic is all that is required since no pressure is exerted on the tissue.

In the selection of an operative method as routine procedure we have been intrigued in the past years by many techniques such as radical excision to the bone crest with curettage of the bone itself, the flap operation, the electro-coagulation method and various methods of medication having for their objective the destruction of the overlying epithelium and connective tissue. That these methods when used with skill and judgment have and do produce satisfactory results there can be little argument. However, observation and experience indicates that no one of these methods may be applied routinely to all cases of periodontoclasia without producing unnecessary tissue loss and often excessive discomfort and pain to the patient. On the other hand, the skillful use of the subgingival curette enters into so many phases of the prevention and elimination of periodontal disease that it may be considered a universal factor in treatment, to be supplemented by the more radical methods when necessity arises rather than superseded by them. When nature's kindly response to the elimination of irritation by careful subgingival curettage is observed, and as skill in the use of the instruments increases there will be less and less need for further operative interference. It is distressing how little knowledge the average recent graduates of our dental colleges have concerning the prevention, to say nothing of the methods of treatment, of periodontal disease. Until such time as the standardized use of subgingival curettage is universally taught, so that the student may observe for himself the tissue reaction, little progress can be made in that direction.

Methods which experience and observation demonstrate to be effective are so simple that their importance is often overlooked. Each stage in the procedure is *exacting in technique,* and experience has shown that herein is the cause of many failures and likewise the reason so many operators become impatient of subgingival curettage, dismiss it as being ineffective, and resort to more radical methods of operation. How often we hear the statement made that a tooth was "scaled" or that a pocket was curetted with no observable results, implying a condemnation or at least a uselessness of subgingival curettage. The point of the whole problem is that of the thoroughness with which the curettage has been performed. Likewise in the matter of correction of traumatic occlusion. A great deal of grinding may be done and still the stability of the tooth not improved or the tortional stress removed. It is again a question of accurate technique painstakingly applied.

A systematic approach to any case of periodontal disease is essential.

First: full mouth radiograms must be had; they are necessary to a study of the supporting tissues of the teeth.

Second: the diagnosis as well as prognosis is made, and if possible teeth beyond the scope of treatment or hopelessly infected are removed prior to the institution of operative procedures.

Third: premedication may often be necessary for the reduction of bacterial growth and inflammation, the result of the very prevalent spirochete and fusiform bacillus of Vincent's.

Fourth: a systematic, forceful and effective means of teaching tooth brushing technique should be set up in every office, and the patient trained by every reasonable means in the correct use of the tooth brush as an instrument for the treatment of periodontal disease and not a toilet accessory.

Fifth: It is essential to correct and to produce as good occlusal balance as possible at this stage. This procedure may be made one of the most valuable and satisfactory in the entire treatment of the case. If properly accomplished, it is a source of the greatest satisfaction to the patient, and there will rarely ever be any question in his mind as to the effectiveness of the result. Whenever there is a feeling of discomfort on the part of the patient, invariably there is something wrong with the technique or the method employed.

Sixth: All of the foregoing has been in the nature of preparation of the field, and we are now ready to begin systematic subgingival curettage. Regardless of the extent of the lesions or their location, one section of the mouth should be selected to begin opera-

tion, and every portion of the gingival tissue about each successive tooth carefully curetted, the depth of the lesion compared to the radiograph and charted on the diagram of the patient's chart. It is not at all uncommon for a skillful operator to complete the curettage of one half of an arch in one sitting of an hour and one-half. This is certainly not "piddling" as subgingival curettage has sometimes been called. As skill increases in this operation it is rarely ever necessary to use an anaesthetic. There is no objection to its use where conditions demand, but experience indicates that the majority of patients are grateful if the treatment can be carried on without an injection. After-pain and discomfort are so rarely present as to be practically negligible. As the respective areas are curetted, note is made upon the diagram as to the extent, depth, and the possibilities of recovery of the deepest or questionable areas. After the initial reaction of the supporting tissues to subgingival curettage, it is a simple matter to determine whether any particular lesions will require further operative interference. This may range from simple recuretting and excision of overlying tissue to occasionally more radical methods. The need for the latter, however, is so rare that their use may be considered almost unnecessary. After following the above procedure we let nature's own forces of repair tell us what should be done to further assist in the restoration of the tissues to health.

Seventh: Periodic prophylaxis as previously discussed is instituted.

Medication and therapeutics has been purposely avoided in this discussion. In the hands of certain individuals there are medicinal agents which act favorably as adjuvants to treatment. It is quite interesting to observe and occasionally experiment with the action of certain drugs where one is convinced that no harmful results may arise from their use. Occasionally agents or methods of application are found which are of value. To say that any of these elements are essential would probably be a mis-statement of fact, therefore, the subject is best left to the discretion of the individual operator.

One of the interesting, and almost universal abnormalities resulting in periodontal disease and destruction is the crowding and consequent overlapping of lower incisors. This condition is undoubtedly due to our racial under-development of the maxillary and mandibular bones. Very few patients are seen where there is adequate space mesiodistally for these teeth, and the crowding often extends to the cuspids. This situation creates one of our most difficult problems in maintaining health of the supporting tissues of these teeth. Usually one or more of the incisors is displaced lingually. These teeth continue to erupt until contact is established with some area in the upper incisors, provided, of course, that the individual maintains the teeth in centric occlusion much of the time. When the mandible is moved into protrusive relation it very often follows that the incisal edge of the displaced tooth is brought into violent occlusion with the upper anteriors thus producing a trauma invariably resulting in the loss of supporting periodontal structure. With the correction by grinding of the incisal relations in these cases, and with the checking of active tissue destruction, even to the point of recalcification of supporting bone, these teeth will often continue to erupt and we see many of them practically exfoliated with a minimum of irritation and inflammation in the periodontal tissues, even when receiving routine care.

The treatment of periodontal disease as we know it today undoubtedly leaves much to be desired, but the same is true of other fields of operative dentistry. Research, observation, and experience are the factors which are responsible for our advances of the present, and they will bear the same relation to the future. The ability to reason simply and think straight are among an individual's greatest assets. The results in the saving of teeth from periodontal disease compare favorably with the efforts of general dentistry in saving teeth from dental caries. The failures, however, come in for more criticism and even cynicism than do the failures in other fields of the profession. This is an unjust attitude which those interested in periodontia should lose no opportunity to correct in the minds of confreres.

Civilization, as we have developed it has wrought many changes and created many demands upon our bodies which we are poorly equipped physiologically, to meet, and we have often used little intelligence in meeting them. We have created a social and economic system whereby it is necessary for

millions of people to labor from dawn to dark in order to obtain the simple essentials of food, shelter, and clothing, and under conditions frequently subversive to health. Our food is determined by taste and catered to by the commercial interests.

Wendell has said: "The life of races is limited just as is the life of individuals. We have already arrived at an advanced stage of racial senility, and that of course goes for all modern civilization. Here and there an individual, perfect in health and physique, holds his own against the devastating forces, but that is because the law of the survival of the fittest sets him apart from the rest."

We can scarcely consider that we have contributed what we should have to human happiness until we have made it possible to provide the fundamental biologic needs of humanity according to nature's plan. No groups are in a better position to teach the above mentioned facts than those of medicine and dentistry, and it should be just as much a part of our plan of treatment as technical procedures.

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Quantitative Analysis of the Various Steps Employed in Periodontal Treatment*

By SAMUEL CHARLES MILLER, D. D. S., F. A. C. D. *and* BENJAMIN B. SEIDLER, A. B., D. D. S.

The present project was undertaken to determine how much average time is spent in performing the various operations required to bring a periodontal case to a successful conclusion. For this, cases were selected at random from the files of the Periodontia Clinic of New York University College of Dentistry. The periodontia work chart and corresponding roentgenographic series were analyzed and the following information was recorded for each case:

1. Name, age, sex, and chart number.
2. Occupation.
3. Subjective complaints.
4. Soft tissue changes (objective signs).
5. Systemic involvements.
6. Total number of erupted teeth (usually obtained from roentgenograms).
7. Number of teeth treated; the difference between this figure and the preceding one was usually due to:
   a. Extraction of teeth with hopeless periodontal prognosis.
   b. Extraction of teeth having carious involvement of pulp.
   c. Extraction to enable a more favorable prosthetic restoration to be made.
8. The total number of visits required to complete the case.

This consisted of:

a. Number of visits, charting
b. Number of visits, scaling
c. Number of visits, toothbrush demonstration
d. Number of visits, curettage
e. Number of visits, equilibration of occlusion
f. Number of visits, miscellaneous procedures including:
   1) Radical procedures: surgery, electrocoagulation and caustics, e. g., silver nitrate and phenol; also, the application of various packs, e. g., zinc oxide and eugenol, guttapercha, etc.
   2) Application of desensitizers, e. g., zinc chloride, formalin, etc.
   3) Sodium sulfide for pocket reduction.
   4) Antiseptics in Vincent's infection, e. g., tincture of metaphen and acriviolet.
   5) Dietary instruction and vitamin prescription.

Each visit ranged from 20 to 60 minutes; the mean was approximately 45 minutes. When two procedures were performed during the same session, a score of one-half was

* Report from the Department of Periodontia, New York University, College of Dentistry.

recorded for each of the steps. Those visits when the patient failed to appear or when no work was done were not recorded. Only those cases in which the treatment was completed were included in the study.

RESULTS OF STUDY

A total of 500 cases was studied. The group was composed of a somewhat greater number of females. There were 307 females to 193 males, corresponding roughly to 3 females to each 2 males. Their ages varied from 12 to 70 years; the mean was computed to be 39.7 years. The greatest concentration of cases lay in the ages of from 30 to 49 years.

TABLE I

Total number of cases studied	500	
Number of males and percentage	193	(38.6%)
Number of females and percentage	307	(61.4%)
Average total number of teeth	23.65	
Average number of teeth treated	20.24	
Average total number of visits	11.88	
Average total number of visits/tooth	.59	
Average number of visits		
Charting	1.52	
Charting/tooth	.075	
Scaling	1.59	
Scaling/tooth	.08	
Toothbrush demonstration	1.17	
Toothbrush demonstration/tooth	.06	
Subgingival curettage	5.19	
Subgingival curettage/tooth	.26	
Equilibration of occlusion	1.94	
Equilibration of occlusion/tooth	.10	
Miscellaneous	.40	
Miscellaneous/tooth	.02	
Number and per cent requiring miscellaneous	126	(25.2%)
Total number of visits		
Sodium sulfide*	74	
Electrocoagulation	6	
Surgery	52	
Splinting (temporary)	19.5	
Diet instruction†	11	
Acriviolet	5.5	
Adam's treatment	2.5	
Silver nitrate	6.5	
Zinc chloride	5	
Formalin	11.5	
Tincture of metaphen	3	
Paraformaldehyde	.5	
Phenol	1	
Zinc oxide and eugenol	6.5	
Guttapercha pack	.5	
	205	

The average total number of teeth was found to be 23.65 while the number of teeth treated was computed to be 20.24. The difference, 3.4 teeth, represents those members recommended for extraction for reasons previously mentioned. The number of condemned teeth is surprisingly low and indicates that routine treatment by students is considered effective for 86% of the teeth presenting for periodontal treatment.

The average total number of visits given each of the 500 cases was computed to be 11.88 which represents .59 visits for each tooth. The entire series of visits usually extended over a period of two to four months.

An average of 1.6 visits was required to chart each mouth. While this procedure bears little direct relationship in effecting a cure by itself, it is important in enabling the operator to have a clear picture of the mouth before instituting treatment. This record included the depth and location of pockets, the objective and subjective signs, and any systemic involvements which might contribute to the existence of the oral aberration. Usually, one-half the visit devoted to charting was allocated to another procedure.

The average number of visits spent in scaling a mouth was computed to be 1.59. This figure represented an average of .08 visits for scaling each tooth. This was the procedure usually performed in conjunction with charting.

An average of 1.17 visits was utilized in demonstrating proper toothbrush technique. This procedure was likewise frequently carried out in conjunction with one of the other operations, usually curettage. The technique most often demonstrated was the Stillman modification of Charters' method. Charters' method was less frequently employed than was interproximal stimulation by means of a small rubber tip.

An average of 5.3 visits was spent in curettage of all teeth, or .26 visits per tooth. This constituted almost one-half of the entire course of treatment. This finding points to the importance of achieving great proficiency in this procedure.

On the other hand, in spite of its importance, an average of only 1.94 visits was utilized for equilibrating the occlusion.

* A computation of the cases in which sodium sulfide was used during the present school year would probably yield much lower results.

† This year, diet studies were made for every patient and advice was given when necessary.

An average of only .40 visits was spent on all the other (miscellaneous) procedures. These operations comprised splinting, radical and conservative surgery, desensitization, sodium sulfide for the reduction of pockets, application of acriviolet and other antiseptics and dietary advice. The exact amount of time devoted to each of these is shown in Table I. Only 126 patients (25.2%) required one or more of these miscellaneous procedures.

SUMMARY

1.—The entire group of 500 cases comprised 39% males and 61% females.

2.—The average age of the patients studied was computed to be 39.7 years.

3.—The average total number of teeth was found to be 23.65 while the number of teeth receiving periodontal treatment was computed to be 20.24.

4.—An average of 11.88 visits was utilized for the entire course of treatment. This was composed of:

a. 1.52 visits for charting
b. 1.59 visits for scaling
c. 1.17 visits for toothbrush demonstration
d. 5.19 visits for curettage
e. 1.94 visits for equilibration of occlusion
f. .40 visits for miscellaneous procedures.

706 Medical Arts Bldg.

Classification and Nomenclature of Periodontal Diseases* (1)

(Based on Pathology, Etiology, and Clinical Picture)

By BALINT ORBAN, M. D., D. D. S. *Chicago, Illinois*

The nomenclature of diseases, pertaining to the supporting tissues of the teeth, is somewhat confused. The reason for this is evident: the different diseases of these tissues have not been classified adequately enough to permit an acceptable nomenclature. Therefore, an attempt is made to classify and name these diseases, giving due consideration to the etiologic, pathologic, and clinical picture. This classification and nomenclature is not to be considered as immutable, but should be flexible enough to allow further changes wherever required. Only by a generally accepted nomenclature can existing misconceptions of diseases of the supporting structures of the teeth be eliminated.

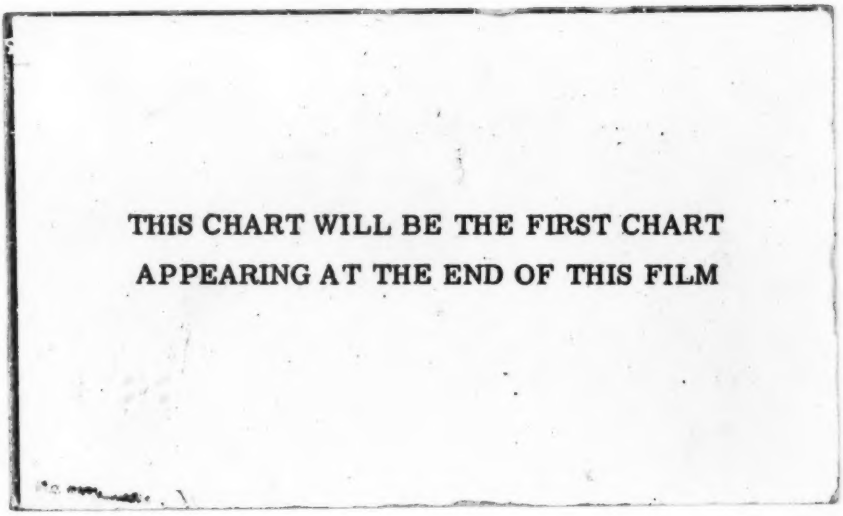
The classification here presented is based on the recommendations of the Nomenclature Committee of the American Academy of Periodontology. Their recommendations were applied to meet practical requirements and, thereby, to correlate pathology, etiology, and the clinical picture. It has been devised with the coöperation of Drs. Harry Lyons, Grace Rogers Spalding, and Arthur H. Merritt. Drs. B. Gottlieb, J. P. Weinmann, and M. K. Hine have also made very constructive suggestions which were carefully considered.

First of all, it should be remembered that the term "periodontium" applies to the supporting tissues of the teeth (periodontal membrane, gingival tissues, alveolar and supporting bone), as was defined by the Nomenclature Committee of the American Academy of Periodontology (1). If the disturbances, encountered in the periodontal tissues, are analyzed from the point of view of pathology,—*inflammation, degeneration, atrophy, hypertrophy,* and *traumatism* can be differentiated.

When *Inflammation*[1] is confined to the gingival margin, it is called "Gingivitis," the suffix "itis" indicating inflammation. If the inflammation penetrates into the deeper structures of the supporting tissues, the term "Periodontitis" is used. In some cases the etiology cannot be determined by clinical or

*From the Foundation for Dental Research of the Chicago College of Dental Surgery.

[1]Inflammation: "complex vascular lymphatic, and local tissue reaction caused by irritation of different kinds." —V. Menkin (2).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laboratory investigations and these must, therefore, be classified as "Idiopathic." Gingival inflammation, from the pathologic viewpoint, may be edematous, serous, or purulent, according to the exudate which forms: ulcerative (Vincent's infection), if the surface becomes necrotic and ulcers form, hemorrhagic, desquamative, or hypertrophic Gingivitis.

In the etiology of Gingivitis local irritation can be distinguished from systemic causes. The accumulation of calculus in the gingival sulcus[1] is a primary factor in local irritation. Faulty technical procedures, food impaction, or other traumatic factors, may also be responsible.

The systemic causes, such as endocrine dysfunction, may be an important factor, especially, in cases of pregnancy and diabetes; acute and chronic infectious diseases frequently cause inflammation of the mucous membranes. (Acute: Scarlet Fever; Diphtheria, Typhoid, etc.—Chronic: Tuberculosis, Syphilis.) A deficient diet may also be the cause for an inflammatory reaction, as in vitamin C deficiency of scurvy, or in vitamin B deficiency. Blood dyscrasias may also manifest themselves by Gingivitis.

In "Periodontitis," the same pathologic pictures appear, extending into the deeper structures. Acute abscess formation is a characteristic feature of this disease when there are deep pockets. The inflammation is always acute in the walls of the gingival pockets and is characterized by the presence of migrating leucocytes. Plasma cells form the characteristic elements of chronic inflammation in the deeper structures. When a marginal inflammation penetrates into the deeper structures, its course follows the capillary lymph routes into the bone marrow and from there into the periodontal membrane (3).

The clinical picture of Gingivitis is identified by swelling, redness and bleeding of the gingival margins, without the formation of deep pockets. In cases of Ulcerative Gingivitis, the gingival margin becomes necrotic and ulcers develop: the tips of the interdental papillae appear to have been cut away. Pain is a characteristic symptom in ulcerative cases. In Inflammatory Hypertrophy, the gingival margin, especially the papillae, are bluish-red and swollen. The inflammation is frequently localized on the labial side and in the anterior region.

In Periodontitis, following Gingivitis ("Period. Simplex"), deep pockets develop and the gingiva, around the pockets, has all signs of inflammation. There is swelling and bluish-redness, due to impaired circulation in the tissues. If acute abscesses develop, the gingival margin is swollen, with a bluish-red discoloration on its surface, accompanied by acute pain. The abscess can break spontaneously through to the surface and pus is discharged. If Periodontitis follows "Periodontosis" ("Period. Complex") local irritating factors, such as calculus, may be absent. However, if the local irritating factor is present this condition may be aggravated by a systemic complication. These deep pockets may develop only on the affected teeth, sometimes, only on one tooth in the jaw. If the teeth show signs of pathologic migration the pocket develops on the side from which the tooth is migrating.

Degeneration[2] plays an important part in the pathogenesis of a specific group of the periodontal diseases. The primary tissue change, in this group, is degeneration of the connective tissue elements in the periodontal membrane, probably accompanied by cessation of cementum formation. Bone resorption follows with considerable widening of the periodontal space. Resorption of the cementum may accompany these changes. The term for this disease group is, according to its pathology, "Periodontosis," "osis" meaning the degenerative character of the disease.

Gottlieb (4) described this disease under the term of "Diffuse Atrophy of the Alveolar Bone." As the disease progresses, the epithelial attachment migrates along the root surface and the gingival pocket deepens. The deep pocket formation is accompanied by a secondary inflammatory reaction. After inflammation has set in reference can be made to Periodontitis Complex, following

[1] "Gingival Sulcus": normal condition.
"Gingival Pocket": pathologic condition.

[2] Degeneration: "That condition of a tissue or an organ in which its vitality has become diminished or perverted; a substitution of a lower for a higher form of structure, either by chemical change of the tissue (true degen.) or the deposit of abnormal matter in the tissues (infiltration)."—Webster.
"A morbid process consisting in the conversion of the elements of a tissue into some inert substance."—Gould.

Periodontosis. The first clinical symptom is migration of the anterior teeth, or loosening of the teeth in the bicuspid or molar region. The difference is due to the fact that a front tooth migrates out of occlusion and can remain firm; whereas a bicuspid or molar, if migrating, causes cuspal interference and, thus, becomes loose. Pocket formation and inflammation are secondary features. The roentgenogram shows extensive bone resorption, localized to one tooth, groups of teeth, or all teeth. The teeth migrate from the diseased area, with deep pockets always developing on the side of the tooth from which migration sets in. The disease frequently attacks adolescent girls, or older men, although no age is excluded in either sex.

Periodontosis often develops in the jaw of caries-free patients. The disease is probably caused by a systemic disturbance, such as diabetes, syphilis, blood dyscrasias, nutritional deficiencies, or endocrine disturbances. The toxic effect of such systemic disturbances may cause a functional insufficiency of the supporting tissues, affecting an impaired physiological regeneration of the periodontal connective tissue which is exposed to mechanical influences (5). The etiology, in a comparatively large number of cases, is unknown (idiopathic).

Atrophy[1] of the gingival and supporting tissues causes recession of the gingivae, exposure of the root surface and resorption of the crest of the alveolar bone. This "Periodontal Atrophy" is frequently due to mechanical stimuli, e. g. excessive or wrong brushing of teeth (6). Atrophy of the supporting tissues may set in at an early age, exposing extensive areas of root surface in some or all teeth. In these cases, no apparent mechanical irritation is present and the cause for the Atrophy is unknown (idiopathic). Atrophy of the gingivae may also follow inflammatory processes during which the epithelial attachment shifts apically along the cementum. After the inflammatory reaction has ceased, the picture of Atrophy becomes clear. Periodontal Atrophy is frequently associated with erosion. With the epithelial attachment constantly advancing in an apical direction, increasingly more root surface is exposed. In advanced age, Periodontal Atrophy is a "normal" occurrence provided the process takes place slowly.

An atrophy of the fibers of the periodontal membrane may set in if a tooth is in *disuse* for a long period of time. The periodontal space then becomes narrow and the cementum hypertrophic. The hypertrophic cementum, as well as the aplastic alveolar bone, lacks Sharpey's fibers; the supporting bone becomes porous. This condition has to be considered as an adaptation of the tissues to changed functional influences. When the functional influences are increased, such as new occlusal stress, a re-adaptation of the tissues can be achieved.

Gingival Hypertrophy[2] is characterized by an overgrowth of the gingival tissues. The hypertrophy may be the result of chronic irritation, similar to the previously discussed group of Inflammatory Disturbances. This type of hypertrophy is not due to an increase of the regular tissue elements but to an accumulation of inflammatory cells and inflammatory products, hyperemic blood vessels, and edema. It has been found that, in actual hypertrophy, the collagenous fiber bundles of the regular connective tissue elements increase in number and size. Sometimes, the epithelium also proliferates abundantly. In many cases of Gingival Hypertrophy the disturbances result in an overgrowth of the fibrous connective tissue (elephantiasis, fibromatosis). The cause of this disease is, in most instances, unknown and, thus, it must be classsified as "Idiopathic."

A specific drug action may be responsible for Gingival Hypertrophy, as in the case of dilantin sodium. This drug is used in the treatment of epilepsy and is believed to produce Gingival Hypertrophy in about 50% of cases. The pathologic picture of the hypertrophic gingivae in the dilantin sodium cases often shows overgrowth of the epithelium with hyperkeratosis (7). The connec-

[1] Atrophy: "A wasting away from want of nourishment."—Webster.
"Diminution in the size, the result of degeneration of the cells or a decrease in the size of the cells."—Gould.
"The wasting away of a tissue, organ or part from disease or defective nutrition."—Dunning.

[2] Hypertrophy: "A condition of overgrowth or excessive development of an organ or part."—Webster.
"An increase in the size of a tissue or organ independent of the general growth of the body."—Gould.

tive tissue elements are likewise enlarged.

Occlusal *Trauma* was frequently, but erroneously, considered to be the sole cause of all periodontal diseases. Extensive investigation (8) has proved that occlusal trauma is a definite cause of certain pathologic disturbances in the periodontal membrane. These pathologic conditions can be summarized in the term *Traumatism.*[1] Trauma may act in vertical, as well as horizontal directions. Vertical overload may not lead to any destructive changes, the strong fiber apparatus of the periodontal membrane being able to compensate the overstress. Horizontal forces, however, result in compression of the tissues of the periodontal membrane, causing destructive changes, such as thrombosis, bleeding and, ultimately, necrosis of the periodontal membrane tissues. Extensive bone resorption follows which, in turn, causes widening of the periodontal space. If Occlusal Trauma acts only in one direction, resorption of bone takes place on the side of pressure, and new bone is formed on the side of tension. If Occlusal Trauma acts in different directions, as in cusp interference, bone destruction takes place all around the root surface due to recurring pressure around the tooth. This causes widening of the periodontal space and loosening of the tooth, thereby impairing mastication. Due to this impaired mastication, soft debris will accumulate on the tooth surface and irritate the soft tissues. Trauma becomes more effective if the tooth loses some of its supporting tissues, due to some periodontal disease, such as Periodontosis or Periodontitis. Even normal occlusal stress may cause traumatism under such conditions. Correction of occlusal relationship, elimination of cusp interference, fixation (splinting) of loose teeth, etc., are the prophylactic, as well as therapeutic measures to be applied in these disturbances.

In *summarizing* this classification and nomenclature a table is presented: in this table the Periodontal Diseases are divided into five groups, according to their pathology: "Inflammation"; "Degeneration"; "Atrophy"; "Hypertrophy"; and "Traumatism." Each one of these groups can be recognized as a separate and independent unit, with the exception of Inflammation. The latter is divided into Gingivitis and Periodontitis: Gingivitis is a primary, Periodontitis a secondary disturbance; Periodontitis follows Gingivitis or Periodontosis.

This classification is based upon Pathology, Etiology and Clinical Picture. The nomenclature is compiled according to this classification, taking into consideration the linguistic requirements. It is hoped that this classification and nomenclature will be adopted for general use, to facilitate understanding between research workers and practitioners. This classification should be changed when new developments require it. It is hoped that the Nomenclature Committee of the American Academy of Periodontology and American Dental Association will not hesitate to propose such changes, or to accept and consider suggestions therefor.

180 North Michigan Ave.

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[1] Traumatism: "Morbid condition of the system due to trauma."—Webster.
"Condition produced by trauma."—Gould.
"The state, condition or injury resulting from trauma."—Dunning.

Some Basic Conditions in Dental Journalism

General Views Matured By Experience and Reflection Through Many Years

By WILLIAM J. GIES

I KNOW . . .

Diseases and their consequences are inevitable, universal, human experiences.

Efforts to prevent or cure, or to alleviate conditions of, disease express noble humanitarian aspirations in health service.

Disorders of the teeth and of their supporting structures are among the most common and disabling maladies, to which dental practitioners equal in number to about one-half that of physicians give professional attention.

Education in preparation for dental practice now includes preliminary cultural study for at least two years in an academic college and graduation from a 4-year professional curriculum in a dental school. This education also prepares the student to continue systematic professional inquiry and reflection—"*graduate* study"—throughout his subsequent career as a dentist.

Development of a worthy dental practice is dependent upon a dentist's ability to grow in professional knowledge and skill, in harmony with the lessons of his increasing experience, and also—in "*graduate* study"—with the current advances in the sciences and arts of oral health-service.

Among the most important agencies for the *graduate* professional education to which dentists should be continually attentive is dental periodical literature.

The periodical literature of dentistry cannot be reliably professional in educational influence unless it is also truly professional in character and import.

I FEEL . . .

Dental journals should voice the soul, express the spirit, elevate the aspirations, and serve the needs, of the dental profession. Dental journals should keep the profession correctly informed and progressively advised.

These sustaining professional functions cannot be performed faithfully, dependably, and effectually, by any journal that is not conducted *primarily* to promote them.

To assure the evolution of dental journalism to the highest degree of dependability for the attainment of these general professional objectives, journals purporting to represent dentistry should be conducted under the auspices of dental organizations that are maintained *primarily* for the promotion of dentistry as a service and for the advancement of dentistry as a profession.

A proprietary journal is published under economic conditions that neither induce nor enable the owner to make the best interests of a profession the *paramount* concern of his periodical.

Proprietary journals, by the adoption of professionally irresponsible advertisement policies, derive incomes sufficient to yield—directly or indirectly—not only large profits for the owners, but also large salaries for the editors.

The self-respect of the dental profession, and of its individual members, cannot be nurtured by journals that are based upon income from advertisements of questionable integrity or propriety.

Societies that publish dental journals should endeavor to acquire the best possible editorial leadership for their periodicals, and should pay honoraria sufficient to enable the editors to give their best service without attendant economic anxiety.

I BELIEVE . . .

A journal published by a dental society should be made fully representative of the professional quality and fidelity of that organization.

A journal of this kind should be given professional, intellectual, and economic support in accord with the society's professional standing.

The editor—competent and representative, and having the confidence and esteem of his colleagues—should be free, within the boundaries of ethical professional responsibility, to guide the development of the journal's policies, to publish his convictions, and to attain the journalistic leadership his abilities and opportunities would promote.

Under these conditions of stimulated endeavor, in the independence of academic freedom —restrained by professional fidelity, journalistic honor and personal integrity—the successive editors could make their journal the educational and inspirational influence their colleagues would wish it to be.

Under such conditions of editorial achievement, aspirants for opportunity in editorial service for dentistry would be encouraged to accept appointments with bona-fide professional journals only, and would decline to serve journals conducted for commercial purposes.

Under such conditions of journalistic excellence, a dental journal purporting to represent dentistry, but conducted primarily to "make money" through advertisement policies of obvious unworthiness or of doubtful respectability, would cease to be acceptable.

Dental journalism, under professional control, is what the editors make it. But editors cannot make dental journals what the best professional interests require until dental journalism becomes a major educational objective, and receives commensurate support from each of the responsible societies.

I SUGGEST . . .

The desire or expectation to receive "something for nothing" is unworthy of the members of a profession or of any other self-respecting group.

Professional journals should be adequately supported by the organizations that publish them; but if such a journal cannot be given the income needed for its creditable development, its discontinuance would not be a loss to the profession.

To sustain a major professional purpose, every dentist should willingly pay the subscription fee, or agree to an apportionment of dues, necessary—in addition to other proper income—for the adequate support and development of the journal that directly represents the dental society of which he is a member.

The evolution of a dental journal, in professional spirit and literary usefulness, cannot be assured by indifferent selections for its editorial service.

Editorial efficiency and achievement, as in all other fields, are—in all but exceptional instances—products of aptitude, attention, understanding, devotion, imagination, *correlated* by industrious endeavor and *matured* by constructive experience.

A staff of editors in which there is selective advancement, at relatively short intervals. to the chief editorship through grades of tested coöperative service—or a prolonged tenure for an editor who, unassisted by a staff, has acquired unusual competence—is a dependable condition for editorial leadership.

Dental journalism will become an increasingly greater educational force, in the evolution of the dental profession, as these three basic conditions are more effectually coördinated to that end: appropriate and adequate *incomes* for maintenance and development; able and devoted *editors*, freed from economic anxieties; interested, critical, and responsive *readers*.

Vincent's Infection (Case Report) *

By ROWE SMITH, D. D. S., *Texarkana, Ark.-Texas*

Male, white. Age 21 years. Plumber and Butane Gas salesman. No serious previous illnesses. Onset with sore throat. S. T. 37 only medication used—prescribed by family physician.

Fourth day: entire oral mucosa, gingivae, hard and soft palate and lips were involved.

Fifth day: patient referred to me by family physician and dentist. (March 19, 1941.)

Medical examination revealed patient acutely ill, toxic, unable to swallow, coughing frequently, with involvement of eyelids and conjuctivae. Temperature 102. Examination otherwise negative except for conditions to be shown in photographs.

LABORATORY DATA

Hemoglobin 90%.
Erythrocytes 5,000,000.
Leucocytes 6,200 with 85% neutrophiles.
Urine negative except trace of albumin.
Blood, Kahn and Laughlen negative.
Smears from mouth and throat showed large numbers of Vincent's organisms.
Smears from eyes negative for all organisms including the gonococcus.

History showed diet included the following each day: Meat at least once. Milk, 1 quart. Green vegetables, average of 2 citrus fruits, tomatoes, and raw cabbage at least once. White bread. Practically no sweets.

Fig. 1—As Case presented. Extensive involvement of entire oral mucosa, gingivae, hard and soft palate, lips and eyelids.

NOTE—The photo engravings in this article were produced from panchromatic negatives made from natural color Kodachromes and are faithful monotone reproductions of the full-color originals. There has been no mechanical or manual exaggeration or retouching of the original copies.

* Read before the American Academy of Periodontology, October, 1941. Houston, Texas.

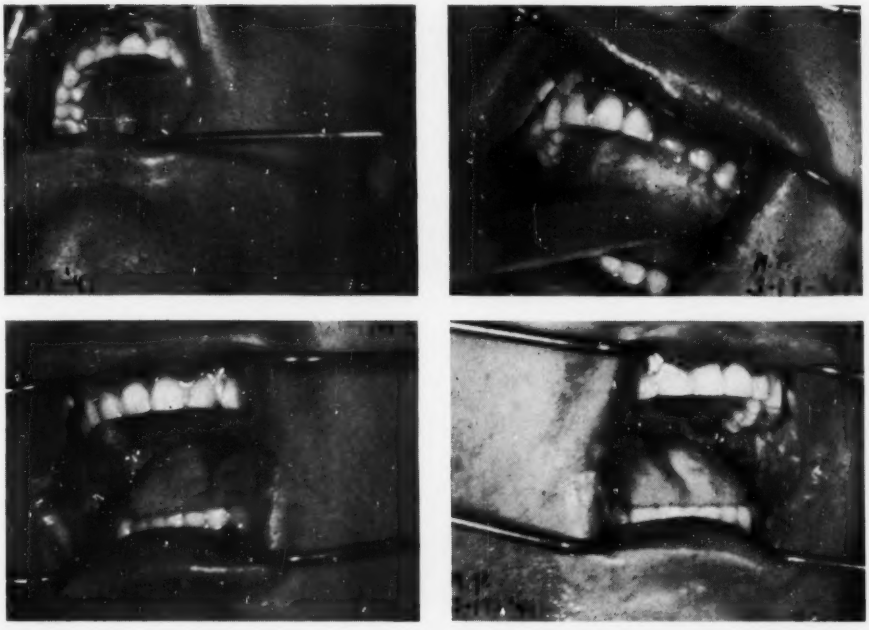

Fig. 2—As Case presented.

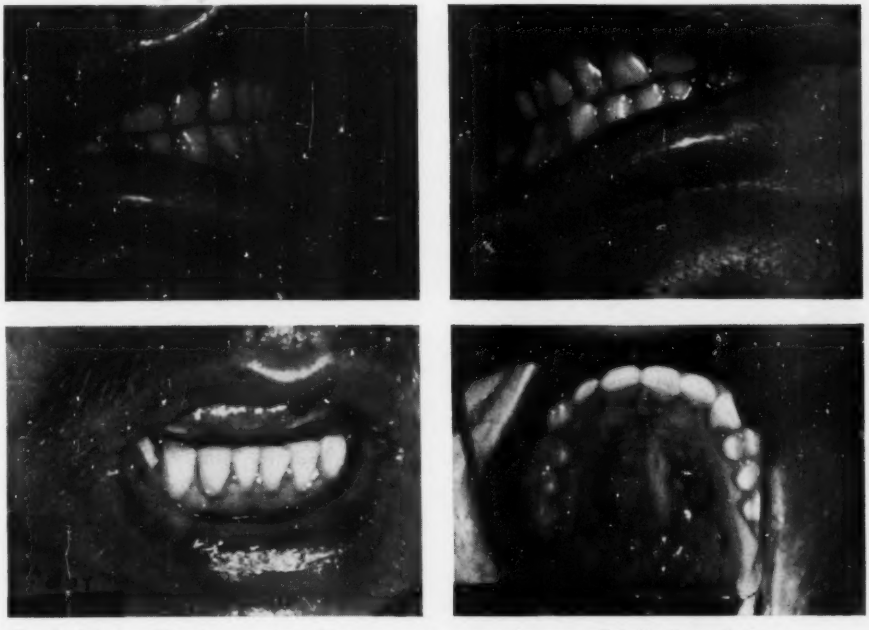

Fig. 3—7th day. Note improvement. Lip area appears worse, however, clinically it was better.

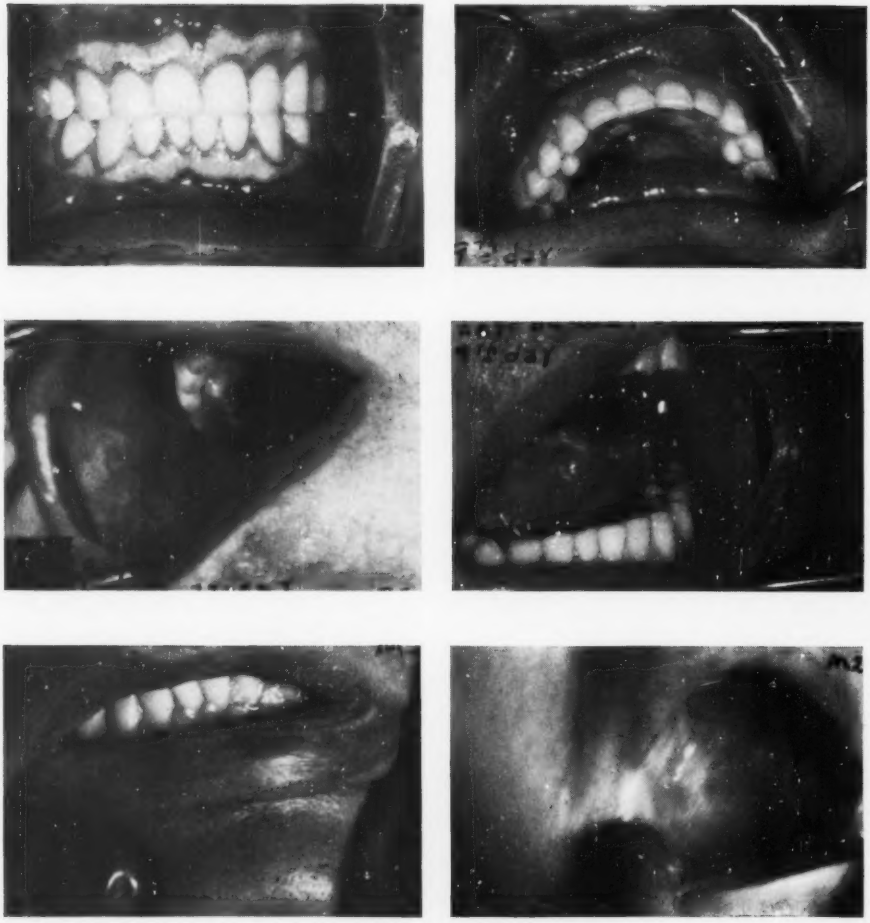

Fig. 4—9th day. Continued improvement.

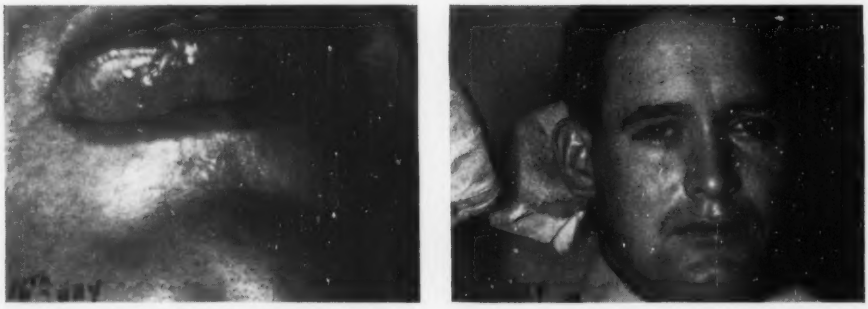

Fig. 5—The usual lingering Vincent's lesion involves the tongue in this case. Note the difference in appearance of patient on presentation and on the 16th day.

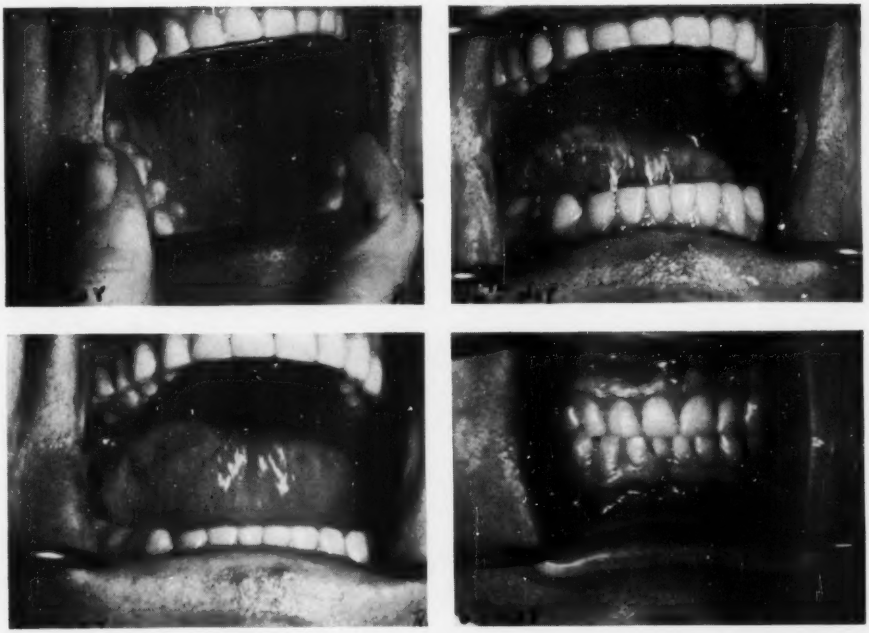

Fig. 6—Same as Fig. 5—16th day.

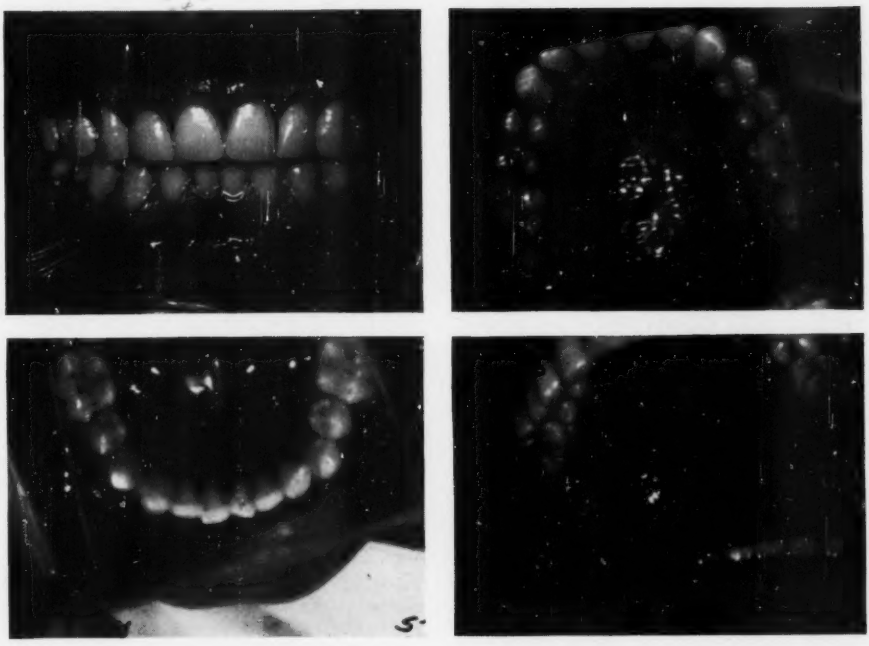

Fig. 7—24th day. All areas completely healed.

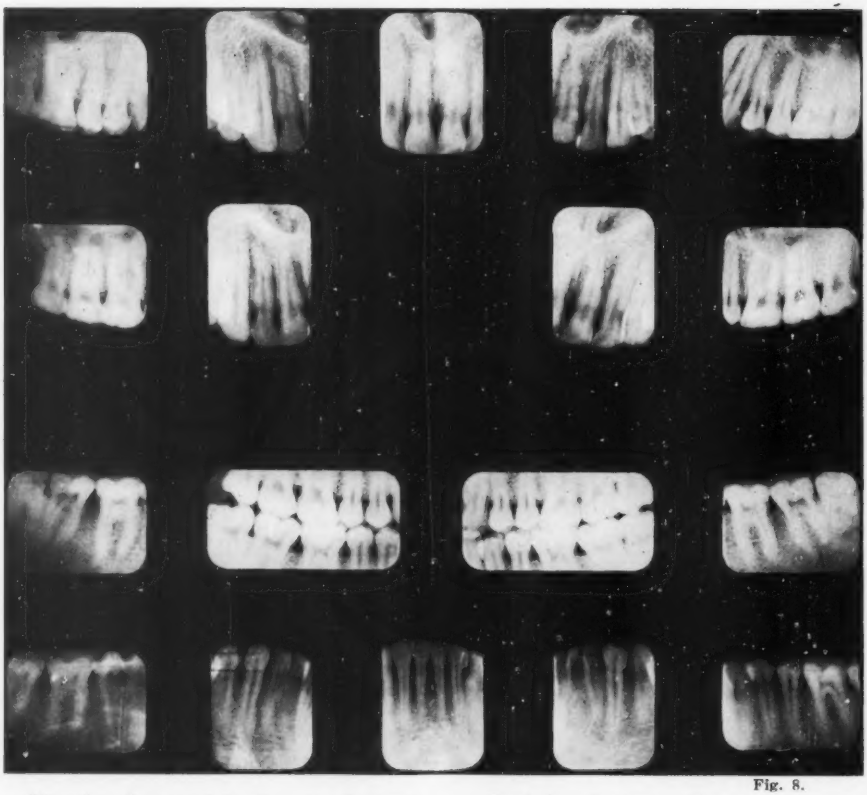

Fig. 8.

No calomel or any injections. 20 cigarettes daily. Alcohol occasionally, mostly beer. Family dental history negative.

As a result of medical examination, pure oxygen (not Dunlop compound gas) therapy was begun. Patient hospitalized with no attempt to control diet. Attending physicians did not advise any other treatment, because of rapid improvement noted morning following first oxygen treatment. Temperature normal in 6 hours. Patient able to take nourishment in 15 hours. Maximum temperature thereafter 99 and remained normal after the 6th day of treatment.

Seventh day. There was involvement of the head and glands of the penis, of the scrotum, and the anus. Same therapy used on these lesions. Smears from other than oral lesions negative. Red spot-like lesions appeared on the upper part of the hips and gluteal region. Did not become necrotic and no membrane formed on these lesions which disappeared in a few days. (Photograph of these lesions on file.)

Dental roentgenographic and clinical examination showed a near normal dentition. No caries was present. No previous dental treatment. Teeth and oral cavity were clean. No prophylaxis was given. No marked bone involvement. (Fig. 8.) Slight clinical pocket between lower incisor teeth cleared up after relief of occlusal trauma.

Fifteenth day: returned to work.

Twenty-fourth day: normal strength and weight regained.

No recurrence after seven months.

SALIENT FEATURES

Suddenness of onset, rapidity of tissue destruction, and extensive involvement of areas not usually attacked by Vincent's infection. Extreme toxicity. No evidence of vitamin deficiency. Pathologic progress seemed to be arrested at once. Sudden disappearance of symptoms of extreme toxicity and rapid repair of all involved tissues. No

other members of family or acquaintances developed similar lesions.

Only therapy used—insufflation and spraying of all areas with pure oxygen gas.

405 East Fifth Street

NOTE—Further comment by the author on the above Case was received by the Editor, May 28, 1942:

"I might tell you that this Case has been shown to numerous medical and dental societies and so far no dentist or physician has ever seen a case like it nor has anyone seen lesions as extensive as these repair so rapidly.

"Dr. Hermann Becks saw this patient personally, examined him carefully, and went over the history of his case with me and the consulting physicians. His only explanation of the case was that it undoubtedly was Vincent's infection, the organisms being of a very virulent type.

"It may be of further interest to the readers of the Journal to report that this month Dr. Balint Orban and I were asked to come to the dental infirmary of one of our large military camps for examination and consultation in regard to five soldiers, age 20 to 26 years, with lesions similar to the ones depicted in this case. The oral and body lesions appeared very similar. The medical histories of all were very similar and also quite like the cases shown here, except each one of the soldier's history reported a previous attack. All of the cases at the camp hospital had been in the isolation ward at the time that we saw them for an average of six weeks and as yet had not yielded to treatment. They had been given the routine treatment for Vincent's infection, fungus infection, etc., plus vitamin therapy. No oxygen gas had been used. It was significant that each of the soldiers came from a different part of the United States, and from different regiments, and none had had any previous contact with each other."

PROGRAM PREPARED FOR THE

Twenty-ninth Annual Meeting

AMERICAN ACADEMY OF PERIODONTOLOGY*

Boston, Mass., August 20, 21, 22, 1942

THURSDAY

9:00 A. M.—Registration—

9:30

First Session of the Twenty-Ninth Annual Meeting. Robert L. Dement, *Chairman*.

REPORTS OF OFFICERS AND COMMITTEES:

Secretary. Raymond E. Johnson.

Treasurer. C. Wesley Marriott.

Committee on Program. Walter H. Scherer.

Committee on Clinics. Edgar D. Coolidge.

Committee on General Arrangements. C. B. Vaughan.

Committee on Scientific Investigations. James A. Sinclair.

Committee of Nomenclature. Harry Lyons.

Committee on Publication. Harold J. Leonard.

Committee on Ethics. Thomas B. Hartzell.

Committee on Credentials. Benjamin Tishler.

Special Committee Reports by: Rowe Smith, C. B. Vaughan, Robert L. Dement, Robert P. Young, Virgil Loeb, Arthur H. Merritt, Konrad Lux, Grace Rogers Spalding, Jesse L. Williams, Willa Yeretsky.

11:00

Presidential Address. Robert L. Dement.

2:00 P. M.

"Rationalization of Periodontal Pocket Therapy." Charles H. M. Williams, Toronto, Ontario.

3:00

"Fungus Infections of the Oral Cavity." T. A. Hardgrove, Fon Du Lac, Wisconsin.

4:00

"Mineral Metabolism and Endocrine Disturbance as They Relate to Periodontal Involvement." Joseph C. Aud, Boston, Massachusetts.

5:00

"What Can Be Done for the Oral Machine in 1942?" E. Melville Quinby, Boston, Mass.

FRIDAY

8:00 A. M.

Past Presidents' Breakfast.

9:00

Report of Past Presidents' meeting. M. Harry Garvin, *Chairman*.

Committee on Scientific Investigation. James A. Sinclair, *Chairman*.

9:15

"The Sulfonamide Compounds in the Chemotherapy of Disease." John A. Kolmer, M. D., Temple University, School of Medicine.

10:15

"The Nutritional Aspect of Paradentopathies." J. Michael Walsh, San Diego, California.

11:15

"Histopathology of Gingival Diseases." H. A. Zander, M. D., Chicago, Illinois.

(Continued on Page 118)

*This meeting has been cancelled for patriotic reasons, but the program is printed for record purposes and to serve as a standard for future meetings of the society.—ED.

Editorials

The Exploitation of Professional Dental Journalism

WHEN in 1919 the *Journal of Dental Research* made its first appearance, it was frankly stated that it was, in effect, "an open break with the past in dental journalism and a move in the direction of complete professional ascendancy in dentistry"; that it would "be neither a supply-house dummy nor an advertising circular" and that it would "endeavor to equal in quality the best of the research journals in the medical and biological sciences."

For more than two decades it has kept faith with that pledge. During those years the *Journal of Dental Research* has been representative of all that is best in dental journalism. That it might continue to uphold the ideals to which it was dedicated, no sacrifice was counted too great by those who appreciated its importance to professional progress. To guard against any lowering of these standards and to aid in its support, the dental profession has undertaken the creation of an endowment fund of $50,000, more than one-half of which has already been paid in cash. It was the thought of the Committee charged with the raising of this Fund, that being able to point to a journal wholly free of commercial influences and to the willingness of the profession to sacrifice itself to provide such a fund, it could then appeal to philanthropic organizations for additional support.

Because of these ideals and the sacrifices which have been made and are being made to maintain them, the opening of the pages of the *Journal of Dental Research* to commercial exploitation has come as a distinct shock to those who have the interest of professional journalism at heart and who have labored long and earnestly in its promotion. That this was done without due consideration as seems probable, does not, unfortunately, alter the situation, nor does it lessen the harm that has been done.

If anything is needed to prove the commercial motive which prompted a manufacturing company to purchase and distribute at its expense, 48,000 copies of the December, 1941, issue of the *Journal of Dental Research,* to members of the dental profession, that proof will be found in the circular letter subsequently sent by this Company to the above mentioned members, calling attention to certain articles published in the December, 1941, issue of the *Journal of Dental Research* dealing with the relative abrasiveness of liquid, *versus* non-liquid, dentifrices.

The publication of these articles in view of the action taken by the Council on Dental Therapeutics, as published in the *Journal of the American Dental Association* for October, 1941, pp. 1682-83, declaring Teel to be "unacceptable for *Accepted Dental Remedies*," makes the action taken by the Committee responsible for selecting research material for the *Journal of Dental Research* well nigh incredible. And when in addition, this same Committee ignored the

recommendation made at Houston in the Report of the Committee on Reprints of the American Association of Dental Editors (of which the *Journal of Dental Research* is a journal-member) that "Both author and editor should in all cases refuse to deliver reprints, and also refuse the privileges of reprinting or circulating any material, to those who seek to profit from its distribution," one finds it exceedingly difficult to understand how such action could have been taken which is so utterly at variance with these recommendations and with every principle of professional journalism.

It has been said of the Christian that if he is to maintain the principles for which he stands, he must not only refrain from evil, but avoid the appearance of it. This applies with equal force to professional journalism.

A. H. M.

In Remembrance

SINCE the beginning of this year, the American Academy of Periodontology has lost four of its members, two of them past-presidents.

Dr. Andrew J. McDonagh, of Toronto, was a charter member and served as President in 1920. He was constructively active in the early years of the organization, and older members recall his many contributions to its development. They, especially, will miss him and his sane counsel.

Dr. William Y. Hayden, of Canada also, had many friends here and his absence will be felt by all.

Dr. L. A. Neil was much loved for his sincerity and devotion to his friends. His fatal illness began just previous to the Houston meeting. Dr. Neil was an enthusiastic pioneer in periodontia in his section of the country and made serious efforts to extend knowledge concerning it.

Word has just been received of the death of Dr. Clyde C. Sherwood an early member and a past-president of the Academy. While in recent years ill health has prevented his regular attendance at meetings his loyalty to the society was continuous; he had hoped to attend the Boston meeting.

The families of these men, well known to many, have our deepest sympathy.

Pacific Coast Paradentic Society

"SELECTED PROCEEDINGS" of the above society (1940-41) and a "Notice" explaining its object were received by the Editor.

> A society formed to promote and advance the science and art of dentistry, especially as it pertains to the prevention and treatment of paradental disease and to promote the study of paradentosis in its entirety; to stimulate the interest of its members in those subjects which are fundamental to a better understanding of paradental diseases; to foster a closer relationship and coöperation between the medical and dental professions in the study and treatment of those diseases; to endeavor to awaken a deeper interest in the subject on the part of the professions and to aid members of the dental profession in acquiring better technic and procedure in treatment.
>
> LESTER E. BREESE, *Secy.*,
> Univ. of California

Considering the serious intent of this society and the high quality of its membership Pacific Coast Dentistry is to be congratulated.

—G. R. S.

ABSTRACTS AND EXTRACTS OF CURRENT DENTAL LITERATURE FOR 1941 (Continued from 13(1) January, 1942)

(c) Miscellaneous Researches

(Continued from 11(2):118. July, 1940)

130—Boyle, P. E.: The effect of various dietary deficiencies on periodontal tissues of the guinea-pig and of man. *J. A. D. A.* **28**:1788-1793. Nov., 1941.

Controlled nutritional studies, utilizing the guinea-pig as an experimental animal, indicates that:

1. Periodontal tissues are not affected specifically by starvation.
2. Vitamin A deficiency causes retardation of bone formation, but does not predispose to gingival infection with pocket formation.
3. Vitamin D deficiency, with a low intake of calcium, causes a typical rickets, with defective calcification of the bones, including alveolar bone.
4. Complete lack of vitamin C causes rupture of the periodontal fibers with hemorrhage; addition of vitamin C brings about rapid repair, with reformation of periodontal fibers.
5. Inadequate intake of vitamin C over relatively long periods results in an inability of periodontal tissues to withstand functional stress, with migration of teeth, which is characteristic of systemic, or diffuse atrophy type of periodontal disease.

Limited clinical and pathologic studies indicate that vitiman C deficiency is a frequent cause of the systemic type of periodontal disease in humans.

—*Martin Blumenfeld*

131—Luethi, H.: Die Vitamin C-Ausscheidung bei gesunden und kranken Kindern. Vitamin C excretion in healthy and sick children. *Z. Vitaminforschung.* **11**:132-153. Nov. 2, 1941.

A study was undertaken to determine exactly how much hypovitaminosis C existed among children of Bern; and, especially, how high a deficit value was found amongst healthy and sick children. The method used was urinalysis of Harris and Ippen, using dichlorphenol-indophenol as an indicator.

75% of the children examined showed a deficit of vitamin C of 0-400 mg., which are normal values. 21% had a deficit of 400-800 mg., which is a slight hypovitaminosis; and only 4% were over 800 mg., which is a severe hypovitaminosis. Hypovitaminosis was especially found in the spring. The average deficiency in April, which is the worst month, was 555 mg.; in August, only 155 mg. Four weeks of holidays in the country caused no alteration in the excretion of vitamin C; only after two or more months' holiday was a higher excretion seen. In tuberculous children, deficit values were found to be higher, and the same was found in children with extensive dental caries.

—*M. B.*

132—McCarthy, F. P.: A clinical and pathologic study of oral diseases. *J. A. M. A.* **116**:16. Jan. 4, 1941.

This article is based on a study of 2300 cases of various oral lesions; the author's primary intention is to give a statistical incidence of diseases and lesions involving the oral cavity, to fill in a hiatus in medical literature. The oral cavity can be considered the "diagnostic mirror" of the body. The dentist should become the diagnostician, and be recognized as a stomatologist.

Dr. McCarthy has found that local irritation due to faulty occlusion, artificial appliances, and dentures is the important factor in production of focal oral lesions. These must be ruled out before considering the rarer causes of oral disease. Endogenous factors producing oral disease must also be recognized by both physician and dentist.

Conclusions:

1. Leucoplakia buccalis is most frequent and important lesion of oral cavity.
2. Vincent's infection is decreasing, and can now be classified as an uncommon oral condition.
3. Gingival lesions are least understood of all oral lesions; great opportunity for research here.
4. Dilantin sodium used for epilepsy causes severe hyperplastic gingivitis.
5. Stomatology should be an important subject in both dental and medical curriculums.

—*M. B.*

133—Miller, S. C., and Seidler, B. B.: **Local microdetermination of glucose in periodontal disease.** *J. D. Res.* **20**:483-488. Oct., 1941.

Ninety-five cases, selected at random, were studied to determine whether alterations occurred in either finger or gingival blood glucose concentrations in periodontal disease. Groups were classified first, according to bone type or speed of alveolar destruction; then, according to the relative amount of bone disintegration; and finally, according to the amount of bleeding produced by the incision.

All of the finger and gingival glucose values had percentage differences between them too close to the limits of experimental error to be significant; charts and tables are used to compare the values found. This study reveals that there is probably no alteration in glucose concentration in periodontal disease, if the method utilized is a tenable one. This is in contrast to changes in phosphatase values, where a definite increase in gingival blood phosphatase, over finger blood phosphatase, has been found in periodontal disease.

—*M. B.*

(d) Etiology

(Continued from 12(2):117. July, 1941)

455—Boyle, P. E.: **Gingival disease.** *Mass. D. Soc. Bul.* **17**:9-13. Jan., 1941.

The author considers periodontal lesions as at least of equal importance with periapical lesions as foci of infection. Infection about the gingivae may cause serious systemic disease; and the prevention or eradication of such lesions is an important step in the elimination of foci of infection. Sterilization of the gingival crevice before extraction of teeth with periodontal infection appears to be an effective way of guarding against serious local and general complications. Maintenance of the normal attachment of the "cuticula dentis" is of great importance in preventive dentistry.

The local type of gingival disease is caused by bacterial invasion of tissues secondary to impaction of food, foreign bodies, or calcified debris about the gingival attachment to the tooth. Endocrine dysfunction and vitamin deficiency are important factors in the systemic type of gingival disease. These factors are discussed, with particular stress on vitamin C. Further developments in endocrinology and nutrition promise means of controlling gingival disease where bacteriologic and immunologic means alone have proved futile.

—*M. B.*

456—Fitzsimmons, L. J.: **Vitamin C deficiency, a cause of periodontal disease.** *J. A. D. A.* **28**:76-80. Jan., 1941.

The author presents recorded data gathered from fifty patients whose average diets were well over on the acid side, which also means that the diets were poor in vitamin C; all were treated for some form of periodontal disease. There is more evidence pointing to the lack of vitamin C as a cause of periodontal involvement than to the acid ash-alkaline ash disbalance as the source of the trouble. He advises a simple mathematical calculation of the acid-alkaline percentage at the beginning of treatment of periodontal disease; it may bring to light a very dangerous overuse of soft non-detergent foods, in addition to the deficiency in vitamin C.

Research has proved that many foods supposedly containing vitamin C are subject to conditions that easily lower the content of ascorbic acid or completely destroy the acid, such as heat, contact with oxygen, preserving process, etc. Complete dependence on a natural source of vitamin C is inadvisable; some form of synthetic ascorbic acid should be prescribed. As laboratory workers have shown that the healing of wounds progresses more rapidly when the vitamin C nutrition is at its height, it is expedient to increase the intake previous to actual operation on the gingivae.

—*M. B.*

457—Franke, Z.: **Acute suppurative periostitis of dental origin.** *Brit. D. J.* **70**:3-10. Jan., 1941.

Periostitis of the upper and lower jaw may originate in three ways: by pulpoapical infection, parodontal infection, or hematogenous infection. In a majority of cases, acute periostitis is preceded by a chronic or subacute inflammation of the dental periosteum, which may be present for years without local clinical symptoms. The initial symptoms of acute periostitis are toothache, sensitivity to percussion, the tooth is looser than the others, and slightly extruded. There is also swelling of the soft parts of the face, fever, and regional lymphadenitis.

Cases vary greatly as to their gravity, depending mainly on the time which has elapsed since the first appearance of the acute symptoms and on the site of the inflammation. Acute periostitis originating from the upper incisors is relatively the mildest, while that originating from the lower molars is usually the gravest. The most dangerous complication of periostitis in the upper jaw is disease of the pterygopalatine fossa. The author describes the various routes of spread of periostitis in both the upper and lower jaws, and the complications that follow. Incision and drainage is the general treatment, with extraction of the involved teeth if acute pulpitis is present.

—*M. B.*

458—Lyons, D. C.: **Caries and gingival lesions due to certain industrial hazards.** *D. Items Int.* **63**:953-954. Oct., 1941.

The author has observed over a period of years, a number of patients working in candy factories and in baking industries. Those in candy factories present the type of caries often described as "juvenile type"—extensive, rapid dental caries, with destruction of considerable tooth structure. They showed no predominance of lactobacillus acidophilus in the mouth flora. The second group, working in the baking industry, showed no increase in caries, but invariably presented themselves with unusual deposits of calculus and soft pabulum covering the teeth. The gums showed a moderate degree of inflammation; in every case, there was rapid destruction of the alveolar process, with the resultant loss of teeth.

Dr. Lyons believes that continual exposure to finely powdered sugar and flour held in suspension in the air in factories, changes the natural mouth flora; this unnatural bacterial condition causes rapid dental caries in one instance, and extensive pyorrhetic lesions in the other. A distinctive type of gum irritation has been observed in workers in a rubber tire factory, but there is not yet sufficient data for a report.

—*M. B.*

459—Rosenblum, L. A., and Jolliffe, N.: **Oral manifestations of vitamin deficiencies.** *J. A. M. A.* **117**:2245-2248. Dec. 27, 1941.

The gastro-intestinal tract occupies a prominent position in the causation of vitamin deficiencies; in addition, it is one of the first systems to present objective clinical signs of vitamin deficiencies. Gastro-intestinal disorders are frequently the cause of deficiency states, due to interference with digestion or absorption. In absence of simple, rapid laboratory tests for vitamin deficiency states, importance of recognizing their oral manifestations is obvious. Lesions of lips, gums, tongue, and buccal mucosa are characteristic of deficiency of vitamin C, and of several members of the B complex; response to specific therapy is shown rapidly by changes in these lesions.

The authors discuss specific lesions due to vitamin C and B-complex deficiencies; of the latter, thiamin deficiency is the only one which produces no known oral lesion. Special emphasis is placed on deficiency of riboflavin and pyridoxine, as the ones which have just recently become known. It is the authors' belief that ariboflavinosis is the most common isolated deficiency that is objectively manifest.

—*M. B.*

460—Sinclair, J. A.: **Vitamin C deficiency.** *D. Items Int.* **63**:313-317. Apr., 1941.

A review of the present knowledge of vitamin C in its function, morphologic effects in deficiencies, and therapeutic uses. McLester says, "the function of vitamin C is concerned in the formation of the intercellular matrix of connective tissue, and its breakdown is manifested in a failure of the colloidal matter to properly 'gel'. The intercellular reticulum and bands of collagen fail to form." Thus vitamin C deficiency may be a factor in clicking, grating, and easy dislocation of the temporo-mandibular articulation by its effect on collagenous fibers of the ligaments, tendons, and fasciae of the joint. It could also be associated with muscular pains in yawning and opening the mouth. Stability, and the ability of tissues of the alveolar ridge to withstand the stress of artificial dentures, seem to be influenced by vitamin C which is often used to relieve sore or tender areas under dentures.

Diffuse atrophy of alveolar bone, in its clinical manifestations and pathology, closely resembles the scorbutic lesions of vitamin C deficiency. This deficiency weakens the collagenous fibers of the periodontal membrane to an extent that physiological stress becomes pathological, resulting in loosening

and pathological wandering of the teeth. Resorption of supporting bone under stress aids the loosening and drifting of the teeth. Mention is made of multiple vitamin deficiencies.

—*M. B.*

461—Sorrin, S.: **Etiology of periodontal disease.** *Mass. D. Soc. Bul.* **17**:19-21. Jan., 1941.

The causes of periodontal disease are numerous and may be due to a combination of factors. S. C. Miller, in a recent article, classifies the etiology as follows:

I. Functional.
  A. Overfunction.
    Occlusal trauma
      1. Excessive stress on teeth.
      2. Insufficient periodontal support.
      3. Too powerful masticatory musculature.
  B. Underfunction.
      1. Non-occlusion.
      2. Premature wear.
  C. Abnormal Habits.
      1. Unilateral mastication.
      2. Abnormal biting habits (biting on foreign objects).

II. Irritational.
  A. Calculus.
      1. Supragingival.
      2. Subgingival.
  B. Improper toothbrushing.
  C. Food impaction.
  D. Effect of improper dental work.
  E. Non-detergent diet.
  F. Mouth breathing.

III. Systemic.
  A. Debilitating diseases.
  B. Dietary and nutritional deficiencies.
  C. Endocrine disturbances.
  D. Allergies and idiosyncrasies manifested in periodontal tissues.
  E. Nervous disorders.

Sorrin enlarges briefly on the factors in this outline. The success of treatment, he concludes, will depend in a large measure on how effectively we remove the factors responsible for the disease.

—*M. B.*

462—Tavenner, R. W. H.: **Vitamin C deficiency and the potato.** *Brit. D. J.* **70**:398-400. June 3, 1941.

Since two-thirds of the food supply of Great Britain has been from overseas, some of the essential elements in the diet may be missing or gravely diminished now. Among the food products affected are those containing vitamin C. Recently at Birmingham Dental Hospital (with which the author is associated), there has been an increase in the number of patients suffering from an acute form of gingivitis leading up to acute ulcerative gingivitis.

The only obvious way to make available more ascorbic acid in the diet—which has definitely been shown to be useful, if not vital, in the treatment of gingivitis—is a wider and more correct use of the potato. If the potato is cooked in the skin, it loses only 10% of the vitamin C content; but if peeled first and boiled slowly, 25% or more is lost into the water. The vitamin C content of properly cooked potatoes is 3.5 mg. per ounce; and 3 oz. of baked potato will yield 210 I. U. Therefore, an increased consumption of adequately cooked potatoes will provide adequate vitamin C in the diet to counteract gingival disease.

—*M. B.*

(e) Symptomatology and Diagnosis

(Continued from 13(1):52. Jan., 1942)

354—Chaikin, B. S.: **Periodontoclasia.** *D. Items Int.* **63**:30-41. Jan., 1941.

A brief history of the recognition and treatment of periodontoclasia is first outlined. The symptoms of the disease are given: traumatic crescents, marginal gingivitis, festooning shortened crests, glossy appearance, mobility, alveolar atrophy, etc. Dr. Chaikin classifies periodontoclasia into two main types; of local origin, and of systemic origin; there is a third type which is a combination of both.

The local type is evidenced by marginal gingivitis, pocket formation, then bone loss, and finally, tooth mobility. It is a result of external forces and operates from without inward. The author lists and discusses the causes, including serumnal and salivary calculi, occlusal trauma, etc. The systemic type progresses in a cycle directly opposite to that of the local. There is first, loosening of the teeth, then bone loss, pocket formation, and finally, gingivitis. It seems to operate from within outward; it is characterized by diffuse atrophy of the alveolar bone from some systemic cause. A marked lack of caries is generally noticeable. Certain diseases have been

cited as causes: diabetes, tuberculosis, late syphilis, and thyroid dysfunction. Also lack of vitamin C.

The histology of the periodontal pocket is given. The treatment depends on correct diagnosis. For the second and third types, a physical examination and treatment are essential. For the local type, determine the underlying cause and remove it. Treatment of the gingivae may be conservative or surgical. The patient must be taught home care of the mouth and he must coöperate, to obtain good results.

—*M. B.*

355—Goldman, H. M.: **Diagnosis and treatment of periodontal diseases.** *Mass. D. Soc. Bul.* **17**:14-19. Jan., 1941.

Examination for periodontal disease should include four parts: dental examination, x-ray examination, systemic examination, and photographs and models. From this data, the diagnosis is made; etiologic factors should be investigated and pathologic changes studied. The author classifies paradentopathies into the following: 1. Gingival abrasion, 2. Senile and pre-senile atrophy, 3. Atrophy of disuse, 4. Atrophy due to occlusal trauma, 5. Gingivitis, 6. Marginal periodontitis, 7. Periodontosis, 8. Syndrome—combination of 6 and 7. Each type is briefly discussed with the etiology and symptoms.

The treatment of periodontal diseases consists of three phases: 1. Correction of etiologic factors, 2. Correction of symptoms present, 3. Institution of procedures for maintenance of health. This includes: removal of faulty dental work, subgingival curettage, surgical elimination of deep pockets, restoration to normal function, adjustment of occlusion, fixation of teeth with splints, and proper home care.

—M. B.

356—Martin, D. J.: **The diagnosis of pyorrhea.** *D. J. Austral.* **13**:201-208. May, 1941.

The author states that only two factors can be relied upon in the diagnosis of pyorrhea:

1. The depth of the gingival crevice and the condition of its base.

2. The radiographic appearance as correlated with the clinical picture.

In regard to the depth, we must bear in mind the variation, within physiological limits, of the position of the gingival attachment to the tooth. Even in crevices 1-6 mm. deep, there may be no true pocket. A true pocket has formed when a definite break has occurred in the crevicular epithelium at the base of the gingival crevice, and not when the crevice exceeds 2 mm. in depth. This may be detected with a fine probe passed completely around the gingival crevice of each tooth.

In the case of the x-ray, it is important to recognize variations, within physiological limits, in the interdental bone. Where there is a slight rarefaction, a true diagnosis of a pocket can only be made by clinical examination of the crevice. Changes in the form and color of the gingivae are to be used as aids in diagnosis, but must not be stressed too much. Crevicular exudate is another factor which must be carefully evaluated; it may be merely serum plus food and cellular debris, or it may be true pus, showing a suppurative process in the soft tissues, while the bone itself may not be infected.

—*M. B.*

357—Ornstein, J. G.: **Gingival manifestations of disease.** *D. Items. Int.* **63**:540-553. June, 1941.

Dr. Ornstein calls the mouth "the gateway for maintenance of life and health"; it often acts as an indicator of maladjustments on the part of the body before, or concomitant with, the appearance of other symptoms. This article considers those factors producing gingival changes, placing the emphasis on differential diagnosis, with little or no mention of treatment.

The subject matter is divided into three broad groups: gingivitis per se; gingival manifestations of blood dyscrasias; and gingival reflections of various body changes and diseases. The diseases discussed are: marginal gingivitis, pyorrhea, Vincent's infection, catarrhal stomatitis, aphthous stomatitis, the leukemias, anemia, purpura, endocrine dysfunction, diabetes, menstrual and pregnancy gingivitis, epulis, Addison's disease, scurvy, pellagra, uremia, and the heavy metal poisonings. Each disease is given a brief review, emphasizing the main diagnostic points in the gingival manifestations.

—*M. B.*

358—Robb, H. M.: **The radiogram in periodontal examination.** *J. Canad. D. A.* **7**:184-185. Apr., 1941.

In general, the radiogram gives an evalua-

tion of the periodontal condition; it also shows conditions other than periodontal that bear on treatment and prognosis. In reading the radiogram, it must be remembered that pockets may be obscured by roots, especially on the labial or lingual; also, a foreshortened ray will make the resorption appear less than it actually is. For this reason a low angle picture of the upper molars is preferred.

The radiogram often shows localized or diffuse alveolar resorption in cases where the outward appearance of the gingivae is normal or nearly so. It also reveals early signs of periodontitis complex and the number, size, direction, and anatomical form of roots; this, correlated with clinical findings, determines the prognosis of many teeth. The appearance of the radiogram is generally the same before and after treatment, so it must always be correlated with clinical findings. A good radiogram is invaluable for a better and more complete understanding of any mouth.

—*M. B.*

359—Scott, T. F. M., and Steigman, A. J.: **Acute infectious gingivo-stomatitis.** *J. A. M. A.* **117**:999. Sept. 20, 1941.

This represents a common group of inflammatory conditions of the mouth among children, about the cause of which there has been considerable doubt. Recent work has indicated that the herpes virus might be the etiological agent. This article represents a study of cases in Children's Hospital, in Philadelphia, in which an attempt was made to:

1. Search for the virus and appearance of its antibody, and thus establish etiology for this condition.

2. Determine clinical picture of proved primary herpetic stomatitis and thus eliminate a series of descriptive names for this disease.

3. Investigate spread of herpes virus in families of patients as an approach to understanding the epidemiology of herpes infection.

The etiology was clarified by isolation of virus of herpes simplex from mouths of patients, and by development of herpes neutralizing antibodies in their blood during convalescence. Clinical picture of an acute systemic infection, characterized by fever, irritability, soreness of mouth, red swollen gums, oral fetor, and regional lymphadenopathy in children is indicative of primary herpetic stomatitis. Herpetic stomatitis appears in two forms: primary, which occurs in young children not previously infected by herpes virus, and secondary, recurrent form, which occurs at any age with little or no constitutional symptoms. Infection spreads readily through susceptible population. Evidence suggests possible sources of endemic foci with special reference to mothers.

—*M. B.*

360—Snell, C. A.: **Early symptoms of periodontal disease.** *J. Canad. D. A.* **7**: 177-179. Apr., 1941.

One of the most important of the early signs is mobility of the teeth; calling the patient's attention to this is helpful in securing his coöperation during treatment. Next, changes in the color of the gingivae must be looked for; digital pressure on the gingivae is a valuable diagnostic aid, but further search for pockets with a suitable probe is of paramount importance. The normal gingival crevice is one to two millimeters, and anything beyond this in depth is to be looked on with suspicion. Many mouths appearing healthy on superficial examination will show deep pockets by the use of the probe.

In treating periodontal disease, curettage must be thorough and subgingival calculus must be painstakingly sought for and removed. The occlusion should be checked from time to time. Bleeding of the gingivae is not to be treated lightly for while usually due to local factors, it may often be a symptom of some systemic disturbance, such as blood dyscrasia, or vitamin C deficiency.

—*M. B.*

361—Tompson, R.: **Periodontal disease: its recognition and prevention by the general practitioner.** *Dent. Digest Prac.* **2**:467-470. Apr., 1942. (Cond. from *D. J. Austral.* Nov., 1941.)

Every dentist should recognize periodontal disease in its earliest stages, and take immediate steps to check it. One of the earliest signs is loss of stippling of the gingivae which become smooth and shiny; they may be normal to bluish-red in color. The x-ray is one of the best means of detecting early signs; thickening of periodontal membrane is the earliest sign, but it may also show condensation of alveolar bone or rarefaction, and hypercementosis. In alveoloclasia, pockets may or may not be present, depending on

whether gum recession has kept pace with bone resorption; the depth of pockets, where present, must be determined. Periodontal disease is usually classed as periodontitis simplex, or horizontal pyorrhea, and periodontitis complex, or vertical pyorrhea; the features of each are discussed. Occlusal trauma is a factor in periodontal disease; clinical and radiographic signs of this are outlined. To prevent inception of the pathologic process, occlusal trauma must be corrected by orthodontics, or by correct grinding; proper operative and prosthetic restorations must be made; calcareous deposits must be removed at regular intervals; home care of the mouth and an adequate diet must be instituted.

—*M. B.*

362—Woods, E. C., and Wallace, S. R. J.: **A case of alveolar atrophy of unknown origin.** *Am. J. Orth. & Oral Surg.* **27**:676-682. Dec., 1941.

Alveolar atrophy is caused by resorption of alveolar bone with insufficient bone regeneration. The case reported is one of alveolar atrophy in a boy, age 10 years. The deciduous teeth erupted normally, but the gingivae were constantly inflamed; between the second and third years, they loosened and were exfoliated. The permanent teeth have erupted normally, but the gingivae have been constantly inflamed and bleed easily. Past history is negative, except for a tendency to develop boils. Family history is negative. Only positive physical findings are tendency to dry skin and nails; all laboratory findings are negative. Oral examination shows normal mucous membrane. Gingiva around anterior teeth and molars is highly inflamed; there is marked periodontoclasia and gingivitis. Teeth are loose and show evidence of migration. Etiology of the condition remains obscure. The condition is still progressing; prognosis is poor, and early loss of all permanent teeth is expected. It is conceivable that the patient has congenital inability to develop alveolar process.

—*M. B.*

(f) Treatment

(Continued from 13(1):60. Jan., 1942)

616—Bradley, G. L.: **Comparison of surgical flap operation and gingivectomy in periodontal diseases.** *D. Digest.* **47**:355-357. Aug., 1941.

Dr. Bradley first discusses briefly the pathology of the periodontal pocket. If conservative treatment fails to eradicate the pockets, the more radical surgical or electrosurgical techniques should be utilized. If the patient is well-educated in home care, approximately the same results will be accomplished by either the surgical flap operation or gingivectomy; the latter resorbs down faster to a zero crevice. Repeated interproximal massage will transform the interdental papillae to a new knife-edge and healthy contour. After inflammation has subsided, the teeth usually become tight and comfortable on mastication; x-rays may even reveal an increase in bone density.

The two operations are described and illustrated with photographs of the various steps. In either case, interdental stimulation with rubber point, and further instruction in home care, after a period of healing, are essential in maintaining healthy resistance in the periodontal tissues.

—*M. B.*

617—Brown, S. and Westmoreland, W.: **The present status of non-surgical treatment of paradentosis.** *Contact Point.* **18**:265-273. May, 1941.

The authors define paradentosis as a chronic, non-specific, expanding disease of the alveolar process, associated with a progressive disintegration or metamorphosis of the paradentium, causing dissolution of osseous elements. The etiology may be divided into local and systemic factors. There are thus two distinct types of paradentosis, due to a difference in etiology; inflammatory and dystrophic types; the symptoms of each are given.

Fundamental non-surgical treatment of paradentosis is instrumentation, which must be carefully performed with suitable instruments. The question of reattachment of paradental tissues to planed root surfaces is still a matter of controversy. Some men suggest that an epithelial solvent such as sodium sulfide be used after scaling. In more advanced cases, radical non-surgical treatment may be necessary to remove gingival tissue and obliterate the pockets. For this, caustic agents, or electro-coagulation, can be employed. No treatment of paradentosis can be successful, however, unless the patient coöperates with proper home care.

—*M. B.*

618—Chapman, E.: **The story of pyorrhea.** *D. Items. Int.* **63**:354-357. Apr., 1941.

Pyorrhea is distinctly a bone disease; it begins with Gingivitis, which if long-continued, finally communicates itself to the underlying bone. When the bone becomes involved, pyorrhea pockets are developed which are incubators for bacteria. It is an insidious condition, resulting not only in loss of teeth, but damaging the whole body and the general health. The x-ray is a good means of checking on the progress of the disease. In later stages, there is a danger of systemic absorption of bacteria. Pyorrhea is much more a source of focal infection than devitalized teeth and yet the latter is of more concern to most dentists. Dr. Chapman strongly urges the education of the dental profession to the importance of treating pyorrhea in its early stages—that is, in the stage of gingivitis; and, subsequently, the education of the public to the need for treatment of gingival conditions.

—*M. B.*

619—Damianovich, J., and Ravizzali, R.: **Nicotinic acid and stomatitis aphthosa.** *J. A. M. A.* **117**:1741. Nov. 15, 1941. (Abstr. from *Arch. Argentinos de Ped.* **16**:1. July, 1941.)

Authors treated buccal aphthae and stomatitis aphthosa by oral administration of nicotinic acid. All but three of fifteen patients were children. Daily dosage of nicotinic acid varied between 75 and 200 mg., divided into three or four doses; tablets were powdered and given in food. Medication was well tolerated; there was no nausea, vomiting, or intestinal cramps. . . . Cure was usually accomplished in three days. There was rapid epithelization of lesions, burning was eased, discharge was diminished, and alimentation normalized. Gingivitis was more refractory and required use of astringents. Vitamin B was not administered.

—*M. B.*

620—Felsher, I. M., and Jones, K. K.: **Prevention of bismuth gingivitis by use of sodium hexametaphosphate.** *J. A. M. A.* **117**:227. July 19, 1941. (Abstr. from *J. Investigative Derm.* **4**:95. Apr., 1941.)

Authors found that hexametaphosphate in fine powder prevented oral uncleanliness, tartar on enamel, carious teeth and roots, and oral foci of infection responsible for bluish pigmentation of gums, or more serious mucous membrane lesions, of patients receiving bismuth therapy. Fifteen parts of it to eighty-five parts of talc was used on toothbrush, and was found to remove calcareous deposits, and food deposits from teeth and gums, preventing formation of mucous plaques. It is not injurious to tissues. Pigmentation of some patients becomes fainter, in spite of continued treatment. . . . About one-half of patients who complained of soreness of gums or mouth were relieved after using powder. There was definite improvement of mouth hygiene in all those who had oral lesions.

—*M. B.*

621—Gottlieb, B.: **Orthodontic treatment of wandering teeth in cases of diffuse atrophy.** *D. Outlook.* **28**:551-552. Dec., 1941.

Firmness of teeth depends on the status of the cementum surface—whether a continual and uninterrupted cementum-apposition is present. In orthodontic treatment, an attempt is made to bring about resorption of alveolar bone and to spare the tooth surface. Dr. Gottlieb reports a case of anteriors which had wandered labially, and which were brought back into line in a few months by means of a denture with a labial arch. At the end of the orthodontic process, the teeth were extremely loose; the wall of the alveoli and the tooth surface were resorbed. The appliance was worn for retention, and the teeth became tight in a few weeks; this was due to reparation by deposition of new layers of cementum. In cases of diffuse atrophy, forcible orthodontic methods should be considered as a therapeutic measure to bring about firmness of wandering teeth.

—*M. B.*

622—Grosby, J. S.: **The flap operation in advanced periodontoclasia.** *Dent., Dig. Prac.* **2**:427-428. Mar., 1942. (Abstr. from *Am. J. Ortho. and Oral Surg.* **27**:612. Nov., 1941.)

The surgical approach to periodontoclasia is not radical, since its aim is to conserve the teeth and bone. Local infiltration anesthesia is used for this operation. A vertical incision is made from the gum crest distal to the canines and extended superior to mucobuccal fold; the interproximal papillae are incised mesiodistally to the depth of the pocket. Labial flap is lifted up with periosteal elevator and the lingual tissue loosened. All granulations are removed with curettes; the alveolar bone is left untouched, or lightly

curetted; root surfaces are curetted and cleaned. Pocket surfaces on flap are gently scraped with scalpel to remove granulations and epithelial lining. When this has been done labially and lingually, flap is replaced and sutured. If there is sufficient interproximal tissue, a single suture is placed through the septa from labial to lingual; if not, the area is allowed to close by granulation. A slight variation is to make a horizontal incision 3 mm. above the gum crest connecting the vertical incisions, which avoids subsequent interproximal suturing and possible resorption of papillae. After closure of the wound, a gauze pad is placed under the lip for several hours for compression; 3 or 4 strips of adhesive are placed on the lip extended across the face, for 12 hours, for immobilization. Ice-bag is applied, and high caloric diet is given. Sutures are removed in 4-5 days. Success depends on correction of other etiological factors.

—*M. B.*

623—Hickman, G.: **Case report.** *Penn. D. J.* **43**:106-107. Apr., 1941.

The patient, a young woman of 28, presented with a complaint of hemorrhage from the gingivae, which was increased on brushing. Examination showed an excellent full complement of teeth, in excellent occlusion. There were moderately deep pockets, and bone resorption, around the posteriors, but no gingival recession; very little calculary deposit was found. She was treated by thorough curettage, and instruction in home care. Over a period of one month, there was no marked change in the condition.

The patient was transferred to another department. Upon questioning, the following was revealed: the patient did not like milk; she had been treated previously for pernicious anemia; she suffered periodically from respiratory infections; the tip of the tongue occasionally became sore, and the calf muscles were often tender. Examination showed a strong tendency toward a raw beefy tongue; the hair was dry and brittle, and the skin excessively dry; symptoms of anorexia, lassitude, and fatigue were found. A diagnosis was made of multiple vitamin deficiency; she also had achlorhydria, and lack of iron intake or assimilation. Patient was put on an acid-ash diet; she was given hydrochloric acid with each meal, and a high dosage of synthetic vitamins A, B complex, and C; liver and iron were also given. In about two weeks, all oral and systemic symptoms cleared up, and the patient felt entirely well. This case illustrates the systemic factors sometimes found underlying periodontal disease.

—*M. B.*

624—Laurin, E. M.: **Treatment of pyorrhea with the help of electrocoagulation.** *J. Canad. D. A.* **8**:121-124. Mar., 1942.

The history of periodontal treatment from ancient times is given. Pyorrhea should be more frequently treated by the general practitioner; odd, complex cases should be referred to a specialist. Diagnosis is simple, based on four common symptoms: color of oral mucosa; pocket formation seen in radiograms; soft, spongy gums, with swelling, and occasionally exudation from pockets; probing of pockets. It is necessary to use several forms of treatment in advanced cases: scaling, surgery, and vitamin C administration. Patient must also coöperate by home care, with toothbrush, etc.

For electrocoagulation, injection or topical anesthesia is used. In deep pockets, electrode must be applied to bottom; where gingiva is hypertrophied, it is applied to surface. There is no bleeding; coagulated tissue sloughs away in 5-6 days, during which time patient must use mouthwash of peroxide or perborate. When slough disappears, scaling is done; then, surgical pack is applied for one week. After this, patient is instructed to use interdental stimulator and toothbrush. Frequent check-ups are necessary during first year. Electrocoagulation should never be used in acute Vincent's infection or acute conditions of pyorrhea; eradicate acute symptoms first.

—*M. B.*

625—Law, L. W.: **Palatal bar fixation of mobile teeth.** *D. Digest.* **47**:494-496. Nov., 1941.

Loss of teeth, and resultant traumatic occlusion, represent the local etiology in periodontia. Lack of balance between masticating force and its direction against the resistance of the supporting tissues results in pressure pericementitis. Infection may be concomitant, or occur subsequent to injury. These drifted, extruded, loosened teeth may be restored to health by means of palatal bar fixation, which prevents continued destruction of the bone surrounding the teeth. The

teeth are stabilized in the mesio-distal axis by ordinary fixed bridgework. A removable palatal bar, anchored by precision attachments in one pontic on each side, immobilizes the teeth in the bucco-lingual plane, with the expectation that firm bone will be rebuilt around the loose teeth. A removable bar has two advantages over a fixed bar: first, it may be removed for cleansing; and second, if bone regeneration results in tightening of the teeth, it can be removed permanently. The technique for its construction is fully described.

—*M. B.*

626—Leabo, W.: **The value of subgingival curettage.** *Bul. N. C. D. Soc.* **25**:67-76. Aug., 1941.

All dental degeneration has fundamentally the same causative factors: improper diet, accompanied by other contributing agencies which are a result of deviation from nature's laws for the maintenance of general health. Irritation of gingival or supporting tissues is the basis of periodontal disease; these can be divided into mechanical, chemical, and bacterial. Elimination of irritation is a most important problem in periodontia. Two of the most universal conditions to be corrected are traumatic occlusion and improper use of the toothbrush.

In all types of operation for prevention and elimination of periodontal lesions, complete removal of calculus is absolutely necessary. Dr. Leabo outlines plan for treatment: full mouth x-rays, diagnosis, treatment for Vincent's infection (where present), correction of traumatic occlusion, toothbrush instruction, and curettage. Surgical elimination of pockets by no means takes place of subgingival curettage; the latter is always necessary in any treatment of periodontal disease. Prophylaxis, as generally practiced today, is entirely inadequate in the prevention of periodontal disease. Thorough subgingival curettage, and attention to other factors involved, are fundamental for true prophylaxis against periodontal disease.

—*M. B.*

627—Lukowsky, E. H.: **Fluorine therapy for exposed dentin and alveolar atrophy.** *J. D. Res.* **20**:649-659. Dec., 1941.

In the first part of the article, dealing with exposed dentin, the problem is to lower permeability, and thereby susceptibility and vulnerability, by forming a surface barrier; in other words, artificial production of a zone similar to natural hypercalcification in vital teeth. Fluorination was used to produce such a barrier; sodium fluoride is readily absorbed by dental hard tissues and by dentinal tubules and basic substance. Fluorine treatment results in partial replacement of the complex $CaCO_3$-$Ca_3(PO_4)_2$, and formation of hard insoluble CaF_2. Sodium fluoride is used in 75% paste applied to dentin exposed to mouth cavity by abrasion, grinding, etc.; for dentin exposed by pulp amputation or extirpation, it is used in 0.7% solution and paste.

In progressive alveolar atrophy, radiographic picture must be properly evaluated before deciding on therapy. Atrophy may take place by decalcification of bone with preservation of basic collagenous substance. To recalcify this collagenous remnant of alveolar bone, the author has used sodium fluoride in oral doses not over 0.01 gm. per day. Sodium fluoride can be classed as a substance with selective curing effect on tissues affected by periodontosis. Fluorine itself is deposited chiefly in teeth and bones, and then in thyroid gland and kidneys; if introduced with lipoids as organic compound, it deposits in cardiac muscle. Retention of fluorine depends on metabolic processes; the lower the rate, the longer the retention. Exact function has not been accurately determined; it has some affect on thyroid gland, reducing intensity of its hormonal activities; it can be supposed that fluorides in tissues act as regulators for cell respiration. Recently, there was support for the view that fluorine specifically contributes to binding of calcium by tissues, especially bone.

190 cases of periodontosis were treated with sodium fluoride. The method was to remove stains from teeth, and prescribe 1% sodium fluoride, 15 drops per day for 30 days; in cases of acute purulent periodontosis; treatment of pockets was added. In all cases of first degree atrophy, teeth were completely strengthened; in second degree cases, atrophy was usually completely cured. (Third degree cases were considered incurable and not treated.) In almost all cases, masticating ability was increased, gingival inflammation disappeared or diminished, and bleeding stopped. Subjective symptoms of itching of gingivae and apprehension in chewing disappeared. In some cases, general

health was improved. Not a single case of fluorine intoxication has been recorded.

—*M. B.*

628—McCall, J. O.: **Treatment of periodontal disease: preventive phase.** *J. A. D. A.* **28**:1303-1308. Aug., 1941.

In seeking the etiology of this condition, it is desirable to omit consideration of inflammation as such, and focus attention on the degenerative processes that precede and also make possible the invasion of microorganisms and the resulting inflammatory changes. The cause of periodontal disease combines certain local elements the nature and action of which are well known, and certain systemic elements some of which are obscure as to their basis; in the majority of cases, local conditions seem to exert the dominant influence. Among the local factors, calculus is probably the most important; then, food impaction, irritation from crown bands, damage to gingivae from improper toothbrushing, etc. These are some of the other factors which tend to cause a type of periodontoclasia called by Box periodontitis simplex. In other cases, traumatogenic occlusion causes alveolar resorption and pericemental degeneration and tends to lead to the suppurative disease called periodontitis complex.

Probably the first type of periodontal disturbance is the inflammation of the gingivae associated with difficult teething; this is usually relieved by increasing the vitamin C intake. Ulcerative stomatitis, and rarely acrodynia, are also observed in infants. In the school child, gingivitis is quite common; this may be the result of vitamin C deficiency of a minor degree, but it may also be caused by other conditions, such as lack of vigorous mastication, etc. Traumatogenic occlusion is another important factor in children, especially in cases of malocclusion.

Preventive phase of the treatment of periodontoclasia in the adult properly begins with the management of the dentition in childhood. Another item in prevention is the factor of vigorous mastication, which stimulates growth of the dental arches, and mills in the teeth to occlusal balance. A third important item is the use of the toothbrush to properly massage the gingivae. Prevention should start in prenatal period by diet, continue through infancy and childhood, and only then is it concerned with the adult.

—*M. B.*

629—Merritt, A. H.: **Periodontoclasia and its treatment.** *J. Ohio D. Soc.* **15**:77-80. May, 1941.

Periodontal disease has been badly neglected in the past, but it is the most destructive disease of the teeth in adult life. Its chief characteristic is bone destruction, beginning at the alveolar crests and continuing toward the apices; coincident with this resorption, pockets of varying depth are formed around the teeth. This destructive process is more frequent in areas of the mouth that are least accessible, and so less well cared for—such as the proximal and lingual surfaces of the molars.

The etiology of periodontoclasia is still largely obscure, partly because of the number of causative factors involved. These factors can be divided into systemic, which are mainly predisposing, and local, which are exciting. Subgingival curettage is the prevailing method of treatment; it requires skill, experience, and proper instruments. Dr. Merritt considers electrocoagulation as a passing fad in periodontia. Surgical treatment, in advanced cases, is designed to eliminate deep pockets. There are three types of operation: the flap operation, the modified flap operation, and gingival resection. The operated area is covered with surgical cement post-operatively, and is left in place, with changes, for two to four weeks. After healing, an effort should be made to balance the occlusion. Post-operative care consists in instructions for proper toothbrush technic, and arrangements for frequent prophylaxis.

—*M. B.*

630—Miller, S. C., and Arvins, A. N.: **Periodontia in internal medicine.** *J. A. M. A.* **116**:2539. May 31, 1941. (Abstr. from *N. Y. State J. Med.* **41**:407-418. Feb. 15, 1942.)

The authors believe that danger of internal infection from periodontal foci is much more potent than that from periapical disease, for following reasons:

1. Much greater zone for absorption involved.
2. Absorption from gingival crevice is more rapid.
3. Resistance to bacterial growth is lower in gingival crevice than elsewhere in oral cavity, due to stagnation and food impaction.

Therefore, comprehensive search for foci of infection should include clinical periodontal examination.

Periodontal disease is primarily dysfunctional in origin; disturbing factors are functional, traumatic, and systemic. Primary therapeutic procedure is restoration of functional coördination of teeth to eliminate strain in function. Dietary deficiencies of systemic diseases, where present, must be corrected. Pus pockets must be eliminated by surgery or subgingival curettage. Etiological factors of periodontal disease are rarely, if ever, single. The disease can be cured, and resulting improvement in health can be maintained.

—*M. B.*

631—Mountain, V. S.: **Methods of treating paradentosis.** *Contact Point.* **18**:204-206. March, 1941.

The author first briefly traces the history of periodontia. At present, there are two main schools of technic; one that follows the teachings of Riggs and Younger and uses subgingival curettage, and another that uses surgical therapy. In the conservative treatment, there is now a tendency to use fewer instruments; the technic of application is the important thing. Surgical treatment includes flap-operation of Zentler, modified flap-operation of Kirkland, and gingivectomy; each is briefly described. Regardless of which technic is used, the essential thing is elimination of all infection, healing of the pockets, and restoration of the tissues to a state of health.

—*M. B.*

632—Ogus, W. I.: **The elimination of the pyorrheal pocket by electrosurgery.** *Am. J. Ortho. & Oral Surg.* Sect. O. S. 135-144. Mar., 1941.

Better cared-for mouths rarely require complete gingivectomy; a combination of radical and conservative methods is sufficient. A study of the osseous structure is more important than pocket depth in determining treatment and prognosis; teeth with unfavorable prognosis should be extracted prior to surgical procedure for pocket eradication around other teeth.

The use of a controlled electric current—the so-called electric knife—is a simple technique for gingivectomy. The electrode substitutes for the scalpel; a surgical pack is unnecessary, because the knife cuts, coagulates, and sterilizes at the same time, creating a sealed bloodclot. Post-operative pain is tolerable, and far less than in scalpel surgery; secondary infection is rare. Only instruments needed are an electrode for cutting, coagulating, and sterilizing, and a dental file to loosen interproximal debris and smooth the interproximal bone. Use of the electric knife must be mastered through practice and experience.

The technique of the operation is described and illustrated. Post-operative care consists in applying a dye daily, and the prescription of a warm mouthwash. When the slough falls off, the final scaling and polishing are done. Satisfactory results are shown by pathologic study of a treated case. The elimination of pyorrheal pockets and complete healing can be effected in one to three weeks.

—*M. B.*

633—Peterson, T. E.: **Immediate elimination of pyorrhea pockets.** *D. Survey.* **17**:1323-1326. Nov., 1941.

The author considers his technic a quick, satisfactory method of treating pyorrhea; a minimum of post-operative care is required, there is very little discomfort, and the end result is the same as with conservative methods. Local anesthesia is used. The depth of the buccal and lingual pockets is marked with a special forceps. These points are connected with a gingival knife, buccal and lingual flaps are removed by blunt dissection, and the diseased interproximal tissue is removed with a curette saw. The exposed root surfaces are thoroughly scaled. The surgical pack used consists of equal parts of zinc oxide and finely powdered resin, mixed with equal parts of eugenol and petrolatum; this protects the gums and controls pain. The pack is removed in ten days to two weeks; a thorough prophylaxis is given, saline mouthwash prescribed, and the patient instructed to use finger massage. Toothbrushing is instituted as soon as possible.

Success depends on observance of the following:

1. The patient must be in good health.
2. There should be adequate bony support for the teeth.
3. Teeth should be fairly firm in their sockets.
4. All occlusal trauma must be eliminated.

—*M. B.*

634—Sand, R. A.: **Periodontal sepsis in relationship to systemic disease.** *J. A. D. A.* **28**:710-714. May, 1941.

In the author's opinion periodontal sepsis is one of the most vicious foci with which

dentists have to deal. Periodontal lesions are of two types, one that has drainage, and the type that does not. Gingival and periodontal lesions harbor an abundant crop of the same organisms found in the tissues proved to be affected by these remote areas of infection.

In the author's experience in association with a group of physicians in a private clinic, the following systemic ailments have been improved or eliminated by the removal of the oral foci; chronic physical exhaustion; vague chronic non-specific gastro-intestinal disturbances; low-grade mild appendicitis, cholecystitis, etc.; cases of arthritis, bursitis, myositis, and dermatoses; cases of subacute bacterial endocarditis; headaches of certain types. In dealing with periodontal sepsis, we must be very selective in the treatment. Curettage often causes repeated flare-ups of systemic symptoms, and is often contra-indicated in certain types of systemic disease which may be due to this focus. In such cases, extraction is often the best treatment.

—*M. B.*

635—Sinclair, J. A.: **Pyorrhea—a non-surgical and non-curettage treatment.** *D. Items Int.* **63**:666-673. July, 1941.

The involvement of an intrinsic factor, if any, in the periodontal lesion depends on the presence of a tooth. The extrinsic factor in this progressive lesion is always an irritant. Phagocytosis fails to remove the irritant in the periodontal lesion, because an *in vivo* state does not exist, due to the anatomical structure of the lesion. The irritant is removed in an unusual way: inflammatory resorption of supporting bone and degeneration of fiber attachments until the tooth is lost either by extraction or exfoliation.

The author discusses the process of repair, and the important role vitamin C plays in this process by maintaining the integrity of the connective tissue elements. In treating paradentosis, the first object is to remove, or render inert, all extrinsic factors without removing the defense elements or paradental fibers. The second object is to reëstablish and strengthen the collagenous white fibers of the paradental membrane. Dr. Sinclair uses repeated packs of a paste of zinc oxide and eugenol, for the first objective; when the root is clearly visible, all foreign irritants are removed and the root polished. Necrotic cementum or dentin is removed with saturated silver nitrate or iron subsulfate solution, and the wound is packed again for one to two weeks. If the gingival sulci are too deep, after a month has been allowed for healing, reduction by surgery is indicated as a prophylactic measure.

—*M. B.*

636—Smith, R.: **Construction of a splint in periodontal treatment.** *D. Digest.* **47**: 118-119. Mar., 1941.

Teeth loosened from extensive bone resorption, or from other causes, can be splinted to firm teeth; this will help control movement, which will aid in eliminating the periodontal inflammation, and in promoting bone regeneration. The splint may be used on any tooth or any number of teeth. Stainless steel 0.012 in. orthodontic wire is used. The technic described is that using a continuous loop, running across the lingual and labial of all teeth to be splinted, and just below the contact point; then, interdental wires are placed and tightened in each interdental space, leaving about one-eighth inch of twisted ends, which are then bent incisally and into the interdental space. After completion of the splint, the teeth should be freed from occlusal stress by careful grinding.

This method of splinting can also be used in the treatment of teeth loosened by traumatic injury; or, to force back into position teeth moved by granulation or proliferating tissue; or, to close up a space in the anteriors caused by drifting, before inserting a fixed restoration.

—*M. B.*

637—Stonés, H. H.: **A technique of scaling and oral hygiene.** *D. Record.* **61**: 225-236. Aug., 1941.

Gingivectomy is advised on all pockets over two mm. in depth; and instrumentation on those less than that. After gingivectomy, calculus that was formerly subgingival becomes supragingival. This obviates the need for various instruments fine enough to penetrate into deep pockets; all that is needed is a set designed to remove visible calculus, and subgingival calculus which is 1-2 mm. below the gingival line. A set consisting of three pairs of scalers, and one thin chisel pattern is fully described and illustrated, as well as methods of holding and using them.

The area to be worked on is painted with antiseptic disclosing solution; scaling is begun with the heavier instruments, gradually

working down to the finer ones at subsequent visits. The visits should be no more than one-half hour duration. Treatment is completed by polishing with a paste, using a polishing cup; inaccessible places are polished with dental floss or wooden toothpicks covered with the paste. For the patient's home care, a detergent diet is recommended and a proper toothbrush technique; the technique described is essentially the Charters method. Two brushes should be used; one with bristles in a concave pattern for facial surfaces, and the other convex for lingual surfaces.

—*M. B.*

638—Walker, R. O.: **Use of nicotinic acid in treatment of oral mucous membrane lesions.** *Dent., Dig. Prac.* **2**: 437-438. Mar., 1942. (Abstr. from *D. Gazette*, **8**:97. Nov., 1941.)

A prepellagrous state is recognized, whose chief oral signs are angry tongue, small pointed, eczematous excoriations at the angle of the mouth, and leucoplakia of the lower lip. Many degrees of this condition exist, but all show the tongue symptom, together with superficial ulceration or patchy hyperemia of the buccal and labial mucosa. Nicotinic acid in 100-150 mg. daily doses is very successful in treating these cases; but failure to produce definite improvement in 72 hours indicates a change of treatment is necessary. The drug has transient toxic effects, which include flushing of the face, tingling sensations, headache, and faintness. These can largely be eliminated by administration with, or after, the main meals, and by one-half hour rest after taking. The author deprecates the use of nicotinic acid for the treatment of lesions other than those of vitamin B_2 deficiency. Administration of the drug should be continued, together with any necessary local measure, until the condition is cleared. Maintenance dose of 50 mg. daily for a further seven days is advised.

—*M. B.*

(h) Vincent's Infection

(Continued from 11(1):52, Jan., 1940)

230—Chaikin, B. S.: **Vincent's infection.** *J. A. D. A.* **28**:1835-1840. Nov., 1941.

Vincent's infection may be transmitted or self-infective; it is generally superimposed on conditions varying from slight marginal gingivitis to advanced periodontoclasia, according to tissue resistance. The many forms of this disease may be generally classified into two groups: acute, and subacute. According to the author, it always starts as the acute form, either mild or severe; but lack of, or insufficient, treatment may allow it to become subacute. The symptoms of both group types are thoroughly discussed. Case histories are presented to illustrate these types.

Practitioners must become familiar with the many forms of Vincent's infection, with their causes and correction. With proper diagnosis and therapy, only painstaking thoroughness and stubborn persistence are necessary for a cure.

—*M. B.*

231—Francis, F. D.: **Vincent's infection.** *J. A. D. A.* **28**:1296-1303. Aug., 1941.

Dr. Francis mentions the status of Vincent's infection during the last World War, when little was known about the disease, and still less about vitamins and hormones. Vincent's infection is known now to assume both an acute and a chronic form, and if the acute form is allowed to run its course it becomes chronic; the chronic form may also become acute. Either form requires nearly the same treatment and home care. The acute form is more easily brought under control, while the chronic form is exceedingly stubborn and requires exhaustive treatment, and even then recurrences may be expected.

The cause of the ulceration is the toxins elaborated by the bacteria involved; the more virulent the organisms, the more toxic are their products, and, of course, the more acute are the symptoms. The extent of damage to the tissues of the host depends on the degree of tissue resistance; the degree of toxicity of the bacterial waste products; the length of time required to reduce the virulence of the organisms, thereby reducing the toxicity. Application of any drug used, together with instruction in home care, constitutes the initial treatment. Some forms of gingivitis—especially gingivitis complicated with fungi infection—simulate chronic Vincent's infection so closely as to defy differentiation.

The status of Vincent's infection today is nearly the same as that of a severe gingivitis of any other etiology. A combination of chronic acid, hydrogen peroxide, and some dye is most satisfactory drug therapy. The procedure is outlined in detail. Copper sulfate is added, if fungus infection is suspected as a complication.

—*M. B.*

232—Hardy, N.: **Vincent's ulceration.** *Brit. D. J.* **70**:401-402. June 3, 1941.

There has been a serious increase in the incidence of Vincent's infection in the industrial areas of Birmingham. At least 90% of cases occur among factory workers, and over 70% of these patients take their meals in canteens. Boiling of all utensils in the canteens for ten minutes is a necessary precaution. Also, the persons affected must be warned of the danger of infecting others.

The majority of cases has been of the virulent type with rapidly spreading ulceration, considerable pain, and slight fever. Bacteriologically, immense numbers of typical Vincent's organisms were found. All cases were segregated; each patient was seen daily until no ulceration remained; each received printed instructions on the necessary precautions against spreading the infection. Standard treatments were used. With very dirty mouths and widespread ulceration, the first treatment consisted of packing with a mixture of zinc oxide, oil of cloves, and cotton wool, left for 24 hours, and repeated three times. The rest of the treatment was local also, and consisted of three variations in application of the standard drugs: 10% chromic acid, H_2O_2, and the dyes; home care was also prescribed. All cases responded within two weeks if attendance was regular; but lapses of two or three days between visits, in the ulcerative stage, nullified previous treatment.

—*M. B.*

233—Kostrubala, J. G.: **Treatment of Vincent's infection.** *Dentistry.* **2**:217-218. Dec., 1941.

It is generally agreed that Vincent's infection is caused by an anaerobic bacillus and a spirochete. The infection is more frequently seen in individuals with low resistance and poor oral hygiene. Any form of medication associated with increasing body resistance and improving the condition of the mouth will bring about desirable results. Vincent's infection may be complicated or followed by infections of deeper structures which cannot be reached by topical drugs; in such cases, the administration of arsenicals has been found to be beneficial. Neoarsphenamine in 0.3 gm. doses is used most frequently for intravenous administration; mapharsen in 0.03 gm. doses is less toxic. Infections caused by cocci complicated by Vincent's organisms are very chronic and resistant to treatment; in these cases, administration of a heavy metal is serviceable as a means of preparing the patient for the arsenical. At least 10 doses of 1 cc. each of bismuth in oil should be given intramuscularly before a course of arsenicals. Intravenous administration of arsenicals constitutes an important adjunct in the management of obstinate cases of Vincent's infection.

—*M. B.*

234—Morrison, J. P.: **A case of Vincent's infection associated with erythema multiforme.** *J. A. D. A.* **28**:1938-1940. Dec., 1941.

Erythema multiforme may be defined as an inflammatory disease of an acute character, characterized by reddish or purplish red, often variegated macules, papules, and tubercles, occasionally becoming vesicular or bullous, and occurring as numerous scattered or grouped lesions of various sizes and shapes. Theories on the etiology vary, but are probably due to some toxic agent or focal infection. The prognosis is good, although recurrence is not uncommon. Treatment consists of internal administration of sodium salicylates; sulfathiazole is of some value. A mouthwash of equal parts of peroxide and water should be used.

Vincent's disease associated with this condition is of infrequent occurrence, and there is very little literature on this association. Dr. Morrison reports a case in detail, showing severe Vincent's infection of the mouth associated with erythema multiforme. According to case reports, the severity of the skin involvement is not necessarily proportional to that of the buccal mucosa. In the particular case reported in this article, the lesions of the oral mucosa were quite severe, while the skin lesions were scattered and mild. In a patient with skin sensitivity, it may be that the action of the toxins liberated from a severe primary Vincent's infection can produce erythema multiforme.

—*M. B.*

235—Schram, W. R.: **Treatment of Vincent's infection.** *Dentistry.* **2**:214-216. Dec., 1941.

The typical lesion of Vincent's infection seems to be produced by a mixture of organisms, mostly spirochetes and fusiform bacilli, in tissue of low vitality. The acute manifestations seem to be initiated in two ways; either by activity of endogenous organisms, or transmission of infection from one person to another. In treatment, it should be

borne in mind that the organisms are anaerobes and partly saprophytic. The logical treatment, therefore, is to expose them to oxygen, and eliminate damaged and necrotic tissue.

Large tartar deposits may be removed at the first visit, if this can be done without injury to underlying tissue. Surgery is contra-indicated in the acute state. The author uses 3% hydrogen peroxide on cotton sponges; the patient should use 1.5% hydrogen peroxide as a mouthwash at home. As the acute symptoms subside, the teeth should be scaled and oral hygiene instituted; all irritations must be removed. Uncomplicated cases are usually brought under control quickly. If no improvement is seen after one or two treatments, systemic complications should be suspected, and laboratory methods of diagnosis resorted to. It is often advisable to refer the patient to a physician for systemic diagnosis and treatment.

—*M. B.*

236—Thoma, K. H.: **Vincent's infection or fusospirochetosis.** *Am. J. Ortho. & Oral Surg.* **27**:479-488. Sept., 1941.

Dr. Thoma classes Vincent's infection as a specific infection, caused by well-known types of organisms, and transferable from one person to another, an infection which often assumes epidemic proportions. Exciting cause is B. fusiformis and Spirillum of Vincent; predisposing causes are of extreme importance, and include vitamin deficiencies, debility, fatigue and blood dyscrasias. Fusospirochetal infection is commonly found associated with agranulocytosis. Local factors are also important, and include lack of oral hygiene, poor restorations, periodontal pockets, and gingival flaps.

Vincent's infection can be divided into acute and subacute forms; symptomatology of each is given. Histopathology and bacteriology are discussed. In treatment, general medication is needed to correct underlying systemic factors, if any. Local drug therapy consists of use of metaphen, methylene blue, chromic acid, or arsphenamine in glycerine, all of which are effective when properly used; oxidizing mouth wash should be prescribed. After disease is under control, scaling and any other periodontal treatment should be given; poor restorations must be corrected, as well as gingival flaps. Adequate diet to be prescribed as listed.

—*M. B.*

237—Webster, J. F.: **Epidemic of ulcerative gingivitis treated at Glasgow Dental Hospital.** *D. Record.* **61**:319-321. Nov., 1941.

The author has observed this condition occurring, formerly, only during the winter months; and now, for the past years of the war, there has been a great increase in its incidence. This suggests to him that it is a deficiency disease, probably due to scarcity of fresh fruit, vegetables, etc., with consequent lack of vitamins. The symptoms are briefly described. Bacteriologically, the tremendous increase of the fusiform bacilli and Vincent's spirochetes over the normal is a diagnostic point.

In the treatment, caustic drugs should be avoided. In the hospital, two solutions are used: 1% sodium ricinoleate, and a mixture of 1% each gentian violet and acriflavine; these solutions are applied one after the other at each visit. Potassium permanganate is supplied as a mouthwash, mainly for its mechanical cleansing. A tonic containing iron perchlorate and potassium chlorate is also prescribed. No surgical work should be done until the condition is cleared up. Patients should be warned of the contagious nature of the disease, and of the proper precautions to be taken.

—*M. B.*

238—Wilkin, F. T.: **An effective sequence in the treatment of Vincent's angina.** *New Zealand D. J.* **37**:73-74. May, 1941.

The author considers that cases that do not respond to treatment are due to re-infection from home sources. Check-up of other members of the family is now asked routinely of Vincent's patients; instructions are given to destroy all toothbrushes, and to sterilize all cooking and eating utensils.

No scaling is done until there has been a good deal of healing of the infection, and the patients are instructed not to use a toothbrush; this is to prevent trauma to infected tissues. Intravenous injection of arsenic, while very beneficial, must always be used in conjunction with local treatment. Oral administration of arsenic in the form of Stovarsol, one-half grain tablets twice a day, can be substituted for intravenous injection. Local therapy consists of swabbing with 4% chromic acid, and a spray of *L. arsenicalis*. In cases where there is marked hypertrophy, a mixture of zinc oxide with 99% oil of cloves plus 1% thymol is made

into a creamy paste, put on long wisps of cotton, and worked down into the pockets. In three days, most cases show shrinkage back to normal.

—*M. B.*

239—Williams, C. H. M.: **Trench mouth—How do you recognize and treat the recurrent condition?** *J. Canad. D. A.* 7:181. Apr., 1941.

The author assumes the recurrent attacks to be subacute. Diagnosis depends on: history, blunting of interdental papillae, marked inflammation of the gingivae, bleeding on touch, lack of characteristic fetor of acute Vincent's infection, absence of visible ulceration, and mild malaise. The use of strong caustic drugs for this subacute condition is not wise. Hygienic conditions which are favorable to continued healing must be established and maintained.

Frequent use of an interproximal cleanser is an important procedure, in these cases; it should be used after each meal and before retiring, for at least the first week of treatment. Additional value is obtained in the early stages by having the patient dip it into hydrogen peroxide before using. The topical application of 2% acriviolet or acriflavine two to three times daily is useful. Two to three packings of the gingival crevices, at 48 hour intervals, are useful in simplifying subgingival scaling. Dietary habits should be investigated, and any systemic treatment necessary should be given. Two to four recall appointments at one to two month intervals are advisable until the papillae have regained their normal form.

—*M. B.*

PROGRAM

(Continued from Page 99)

12:30 P. M.

Luncheon—Academy Round Table. Rowe Smith and Walter Leabo, *Leaders.*

1. "Aims and Objectives of Dental Education." Leroy M. S. Miner, M. D., D. M. D.
2. "Oxygen, a Therapeutic Agent Which Can Influence the Character of Periodontal Tissue." Balint Orban.
3. "The Value of a Complete Dental Roentgenographic Examination in Regard to Periodontal Disease, Especially as to Diagnosis and Treatment." Edward Ball.
4. "Restorative Dentistry, Its Value in Establishment and Maintenance of Periodontal Health." Ralph C. Cooley.
5. "The Value of the Dental Hygienist to the Periodontist." Juanita Wade.
6. "Oral Manifestations of Fungus Infection." T. A. Hardgrove.
7. "Vincent's Infection." Raymond E. Johnson.
8. "Instrumentology in Periodontal Treatment." Justin D. Towner.
9. "Systemic Disturbances as an Etiologic Factor in Periodontal Disease." Celia Rich.
10. "Value of Kodachrome for Diagnosis and Pre- and Post-operative Periodontal Records." Olin Kirkland.
11. Ladies' Group. Mrs. Robert L. Dement. (Subject to be announced.)

P. M.

CLINICS. Edward D. Coolidge, *Chairman.*

"The Relation of Vitamin Deficiencies to Periodontal Diseases." Paul E. Boyle, Boston, Mass.

"Periodontal and Oral Disease." (Illustrated with kodachrome and roentgenographic slides and models.) Rowe Smith, Texarkana, Texas.

"Clinical Periodontal Lesions." (Illustrated by hand-painted moulages.) Bernard S. Chaikin, Boston, Mass.

"X-Ray in Periodontal Examinations." Arthur P. Young, Boston, Mass.

"The Importance of Diagnosis in Restorative Dentistry." F. Raymond Garvey, St. Paul, Minnesota.

"Case Management in Periodontia." A. Daiht, Boston, Mass.

"A Simple Stress-braking and Splinting Device for Use in Partial Dentures." George Stein, New York City.

"The Use of Oxygen, Paraformaldehyde, and Silver Nitrate in the Treatment of the Gingival Pocket." Balint Organ, Chicago, Illinois.

"Surgical Treatment of Periodontoclasia." Clark E. Chamberlain, Peoria, Ill.

"Kodachrome Clinic of Mouth Lesions." Kurt H. Thoma, Boston, Mass.

7:30—Annual Banquet and Dinner Dance.

SATURDAY

9:00 A. M.

"Tyrothricin with Special Reference to its Use in Sinus Disease." Joseph L. Goldman, and Emanuel B. Schoenbach, Mt. Sinai Hospital, N. Y.

9:45

Incidents of Office Practice.

"Hyperplasia of the Gums Due to Dilantin Therapy." Edward L. Ball, Cincinnati, Ohio.

"A Diffuse Atrophy Case of Periodontoclasia." Arthur H. Merritt, New York City.

"Gingival Manifestations of Acute Monocytic Leukemia." Carlos H. Schott, Cincinnati, Ohio.

"Caries Prevention in Periodontia." R. L. Wescott, Evanston, Ill.

"Report on a Survey of Tooth Brushing Habits." Isador Hirschfeld, New York City.

"Tuberculosis in the Mouth and Throat as Seen by the Dentist." Frederick Vastine, Shamokin, Pa.

11:15

"Canada's Eastern Arctic, Its Eskimo People and Their Periodontal Diseases." Motion Film. Charles H. M. Williams, Toronto, Ontario.

OFFICERS—1941-42

Robert L. Dement, President, Atlanta, Georgia.
Samuel R. Parks, President-Elect, Dallas, Texas.
Raymond E. Johnson, Secretary, Saint Paul, Minn.
C. Wesley Marriott, Treasurer, Cleveland, Ohio.

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PERIODONTAL DISTU

Classification and Nomenclature on the Basis of Patho

By B. ORBAN, M.D., D.D.S., (
(Foundation for Dental Research of the Chicago

PATHOLOGY	INFLAMMATION				DEGENERATION	
NENCLATURE	GINGIVITIS		PERIODONTITIS		PERIODONTOSIS	
	LOCAL	SYSTEMIC	SIMPLEX	COMPLEX		
ETIOLOGY	Calculus Food impaction Irritating restorations Drug action Etc.	Pregnancy Diabetes Other endocrine dysfunctions Tuberculosis Syphilis Nutritional disturbances Drug action Allergy Hereditary Idiopathic Etc.	Secondary to Gingivitis Etiologic factors similar to Gingivitis	Secondary to Periodontosis Etiologic factors similar to Periodontitis	1. Systemic disturbances a. Diabetes b. endocrine dysfunctions c. Blood dyscrasias d. Nutritional disturbances e. Nervous disorders f. Infectious diseases (acute & chronic) 2. Hereditary 3. Idiopathic	1 2 3 4 5 6
PATHOLOGY	Acute or chronic inflammation localized at gingival margin Can be: a. Edematous b. Serous c. Purulent d. Ulcerative (Vincent's) e. Hemorrhagic f. Desquamative g. Hypertrophic h. Etc.		Chronic inflammation of gingiva progressing into bone causing resorption of supporting structures. Acute abscess formation in deep pockets. Acute inflammation close to wall of pockets, migration of leucocytes through wall of pocket.		Degeneration of the connective tissue elements in the periodontal membrane. Bone resorption. Widening of the periodontal space. Migration of epithelial attachment. Hypertrophy of epithelial rests. Pocket formation.	S g a o c i I p o s
CLINICAL PICTURE	Manifold according to pathology. Swelling and bleeding of gingivae. Change of color from pale pink to red or bluish red. Pocket formation slight.		Deep pockets on all or single teeth depending on the case. In acute abscess formation extensive pain. In Simplex cases much calculus. In Complex cases little calculus, if any.		Migration of teeth. Formation of diastemas. Loosening of teeth. Often caries immunity. As a rule attacks young girls and older men. Pocket formation a late symptom (Periodontitis complex). There are cases where all teeth are attacked and cases where only one or some teeth are affected.	E o T G t e o r

DISTURBANCES

of Pathology, Etiology and Clinical Picture.

.D., D.D.S., *Chicago*
he Chicago College of Dental Surgery)

	ATROPHY	HYPERTROPHY	TRAUMATISM
	PERIODONTAL ATROPHY	GINGIVAL HYPERTROPHY	PERIODONTAL TRAUMATISM
enoces - ias is- ders seases onic)	1. Local trauma (toothbrush) 2. Presenile 3. Senile 4. Disuse 5. Following inflammation 6. Idiopathic	1. Chronic irritation (see inflammation) 2. Drug action (Dilantin sodium) 3. Idiopathic (Gingivoma, Elephantiasis, Fibromatosis)	Occlusal trauma
le- ontal orp- the Mi- al rophy .	Simultaneous atrophy of gingival margin and alveolar bone. Recession of gingiva, exposure of cementum surface. No inflammation; no pockets. In disuse, atrophy of periodontium sets in with osteoporosis in the supporting bone.	In most idiopathic cases overgrowth of fibrous connective tissue of gingiva (fibromatosis, elephatiasis gingivae). Accumulation of inflammatory elements in cases of inflammatory origin. In dilantin sodium cases also extensive epithelial growth.	Compression of periodontal membrane. Thrombosis, necrosis, bone resorption on side of pressure. Stress on fibers of tension side. Widening of periodontal membrane as the result of functional adaptation. If trauma acts in axis of tooth, fiber attachment becomes stronger and bone more dense.
mas. ty. oung . late tis e cases at- ere eth	Exposure of root surface on some or all teeth. Thermal sensitiveness. Gingiva mostly atrophic, thin, pale. In disuse, extrusion of the tooth occurs without gingival recession.	Overgrowth of gingiva often covering entire crown of teeth. If inflammation present, bleeding; if missing, gingiva tough, not bleeding. Sometimes localized to papillae, other times generalized.	Cusp interference leads to loosening of teeth due to widening of periodontal space. Sensitiveness to percussion, sometimes to thermal changes. If trauma is in one direction, tooth may move out of occlusion.